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STANDARDS OF BASAL METABOLISM IN NORMAL INFANTS AND CHILDREN *

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The advent of less complex methods of determining the basal metabolism of adults has made it possible for a large number of investigators to enter this field of research. The fact that such determinations have been of distinct clinical value in some cases has been shown by the works of DuBois, Means, Boothby and others, and, as a result, many clinicians in hospitals throughout the country are now using the small portable apparatus in their investigations.

Until recently little has been done on the metabolism of infancy and childhood, although occasionally investigators in the adult field have studied the basal metabolism in obscure conditions of childhood. Up to date, however, there has been no normal standard with which to compare these studies, and, as a result, it has been impossible to interpret these findings.

The purpose of this paper is to present in a concise form the basal metabolism curves for infancy and childhood which may be used as standards for comparison by future investigators.

In order to establish the normal basal metabolism of infants and children, a series of investigations by Benedict and Talbot on 258 normal infants and children were carried on over a period of ten years and recently have been reported.[1] These studies have been charted and smoothed curves drawn to show the average trend of the metabolism. A line representing 10 per cent. deviation has been drawn on either side of the average curve to delineate what may be considered a normal variation from the average.

Charts 1 to 6 represent the basal metabolism of normal infants for the first two years of life, and may be used for comparison in studying the metabolism of pathological cases up to two years of age. During this early period of life the changes in the metabolism are so great that the results have been plotted in months rather than in yearly periods.

* Submitted for publication, March 29, 1921.

1. Benedict and Talbot: Metabolism and Growth from Birth to Puberty, Carnegie Institution of Washington, Publication No. 302, 1921.

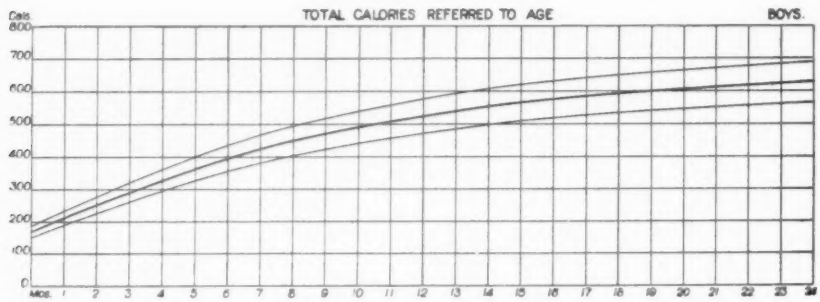

Chart 1.—Basal metabolism of boys during first twenty-four months of age, showing the total calories per twenty-four hours.

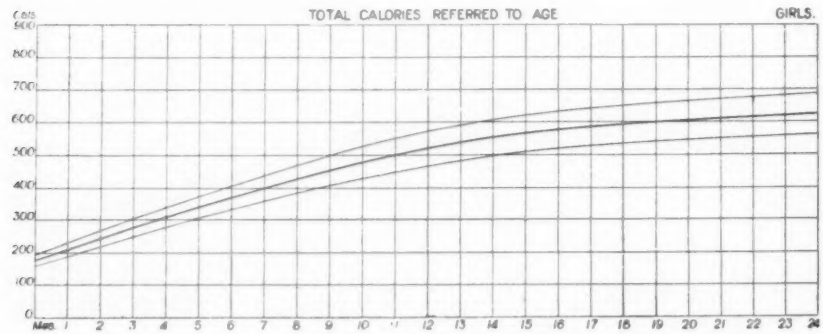

Chart 2.—Basal metabolism of girls during first twenty-four months of age, showing the total calories per twenty-four hours.

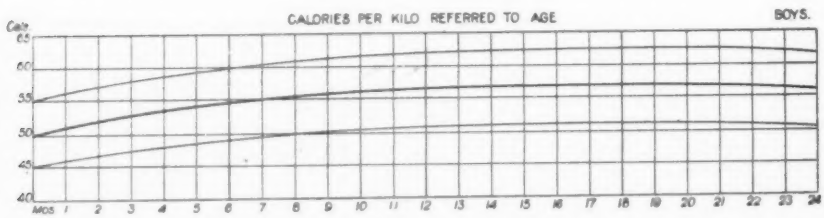

Chart 3.—Basal metabolism of boys, calories per kilogram of body weight per twenty-four hours during the first twenty-four months of age.

The metabolism of the new-born infant, that is during the first few days of life, requires special standards. The average metabolism per twenty-four hours is as follows:

Total = 142 calories; per kilogram of body weight, 42 calories, and per square meter of body surface 612 calories.[2] The metabolism of the new-born infant may be computed very closely by the formula of Benedict and Talbot.[3]

A comparison of the basal metabolism charted in months with that charted in years shows that the curves on the two charts are identical; the former being stretched out to such a degree that the curves are less abrupt and more easily readable.

Charts 7 to 14 show the curves of the basal metabolism of children from birth to puberty, and include the age periods given in Charts 1 to 6, as well as later childhood. As in the first charts, the area of 10 per cent. deviation is shown by lines on either side of the average curve. The metabolism during puberty will have to be studied further before any standards may be accepted, and the age periods on these charts after 12 years should be taken only as temporary suggestions.[4]

If an investigator wishes to study cases from the point of view of total calories correlated to weight only, the multiple prediction formula [5] may be used. This formula does not make it possible to study the metabolism per unit of weight or per unit of body surface without further computation.

Qualifications necessary for basal metabolism.—To determine the basal metabolism of adults it is necessary for the patient to be in the "postabsorptive state," which occurs about twelve hours after the last meal, to have a normal temperature, and to be in absolute physical repose. In infancy, on the other hand, the determination of the basal metabolism is complicated by the fact that the infant must be fed in order to keep it quiet over the necessary period. The rise in metabolism due to restlessness and crying is so great that it overbalances the increase in metabolism due to the "stimulating action" of food; the

2. Benedict and Talbot: The Physiology of the New-Born Infant, Carnegie Institution of Washington, Publication No. 233, 1915.

3. Total calories $= 1 \times 12.65 \times 10.3\sqrt[3]{wt^2}$. In this formula 1 = length, 12.65 = constant, $10.3\sqrt[3]{wt^2}$ = Lissauer's formula for body surface.

4. Papers by F. G. Benedict and M. F. Hendry on the Basal Metabolism of Girl Scouts are appearing in the Boston M. & S. J. **184**:217, 282, 297 (March) 1921.

5. Benedict: Proc. Nat. Acad. Sc. **6**:7, 1920, applied the multiple prediction formula which is derived by biometric analysis of the basal metabolism of 136 men by himself and Dr. Harris to the boys in the Benedict and Talbot Series. The formula: $h = 66.4730 + 13.7516w + 5.003s - 6.7550a$. In this formula h = total heat production per 24 hours, w = weight per kilogram, s = stature in centimeters, a = age in years. It was found that the metabolism of boys who weighed above 10 kg. could be predicted with an average deviation from the individual prediction of ± 56 calories or 6.3 per cent.

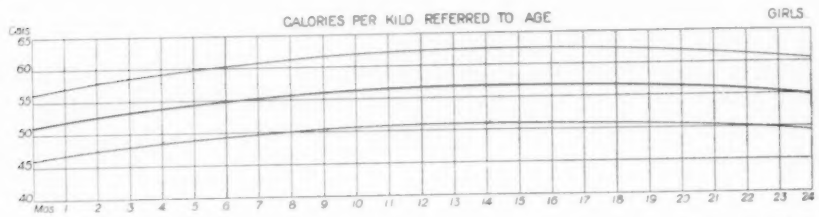

Chart 4.—Basal metabolism of girls, calories per kilogram of body weight per twenty-four hours during the first twenty-four months of age.

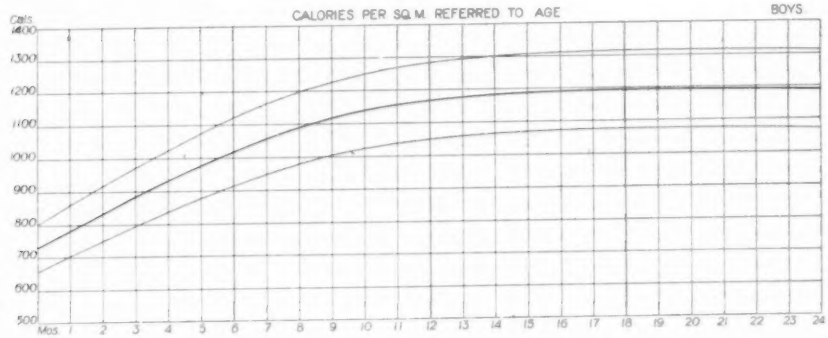

Chart 5.—Basal metabolism, calories per square meter of body surface per twenty-four hours of boys during the first twenty-four months of age.

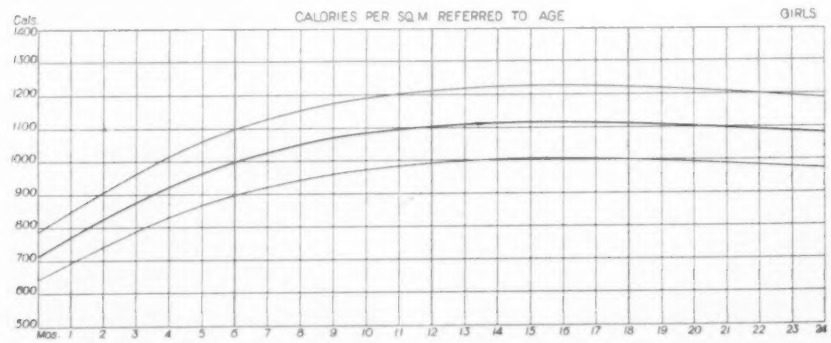

Chart 6.—Basal metabolism, calories per square meter of body surface per twenty-four hours of girls during the first twenty-four months of age.

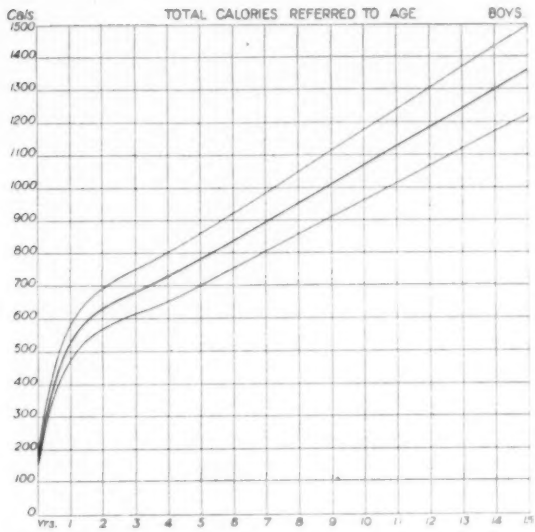

Chart 7.—Basal metabolism, total calories per twenty-four hours of boys by years. The curve is projected from 12 years onward.

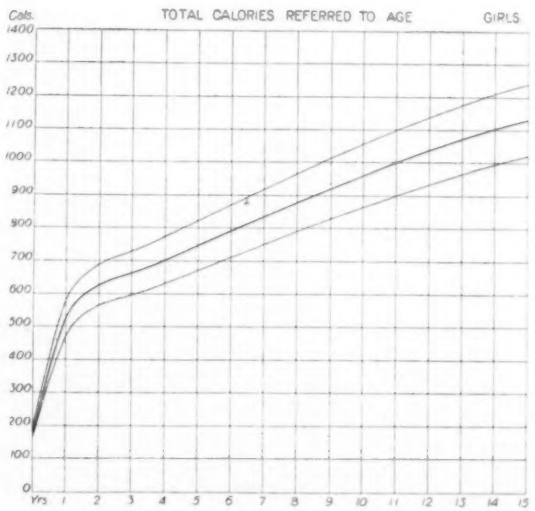

Chart 8.—Basal metabolism, total calories per twenty-four hours of girls by years. The curve is projected from 12 years onward.

increase in basal metabolism due to the "stimulating action" of food being from 9 to 14 per cent., and that due to muscular activity often from 30 to 40 per cent., and occasionally more than 100 per cent. The curve of the "basal metabolism" during this age period is higher than absolute basal figures because of the "stimulating action" of the food. On the other hand, experience has shown that the basal figures obtained after the ingestion of food are usually lower than figures that might be obtained during fasting. The basal metabolism, therefore, during the first two years of life may be more closely approximated with food in the stomach than during starvation, and these curves may be taken, to all intents and purposes, as standards with which to compare the metabolism of infants studied under identical conditions.

To determine the basal metabolism of children over 2 years of age, it is essential that the patient be in absolute physical repose, without food in the stomach, and, if possible, asleep. In older children, however, it is more difficult to obtain sleep.

Determinations of the basal metabolism of infancy and childhood have been made for the most part in a respiratory chamber. The technical difficulties involved by the use of this apparatus, however, make the determinations both difficult and expensive. Investigations made with the portable apparatus, on the other hand, are simpler, but since this apparatus requires the use of a mouthpiece and noseclip, it is unsuited for work with children. The only practical apparatus for determining the basal metabolism of children, therefore, is the respiratory chamber, and it is probable that this is the only method which will be practical for children up to 12 years of age. All so-called basal determinations with an apparatus requiring the use of a mouthpiece and noseclip should be looked on with suspicion until the results have been checked up with those obtained on the same child in a respiratory chamber.

Studies of the basal metabolism have shown that sex influences the height of the basal curve in children. These differences have been chartered and published.[6] The variation due to sex is less marked in infancy than in later childhood, but enough variation is found during this period to warrant the plotting of separate charts for each sex.

Basal metabolism determinations may be made in any well fitted up laboratory suited for this purpose. The great difficulty lies in the correct interpretation of the results. It is necessary to know what are the normal variations of metabolism, and what factors lead to these variations. This would be very simple if all normal children of a given age were of the same weight, height and physical conformity. If this were so, their basal metabolism would, in all probability, fall within 6 per cent. of the average curve. The physical development of grow-

6. Carnegie Institution, Washington, Publication No. 302, pp. 179-181.

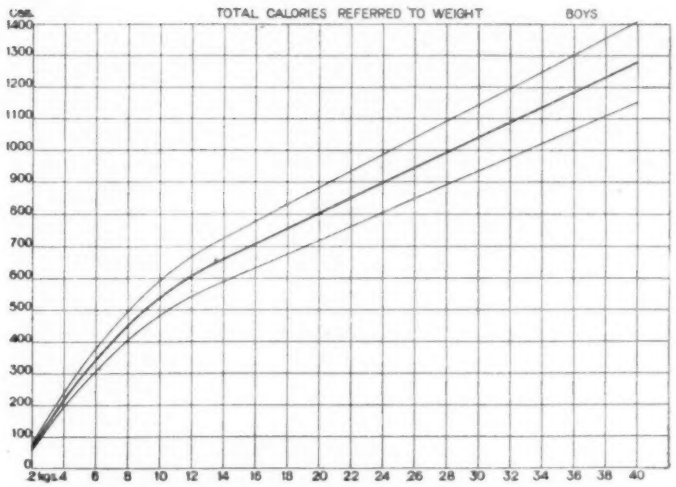

Chart 9.—Basal metabolism of boys, total calories in twenty-four hours at different weights. The curve is projected from 32 kg. upward.

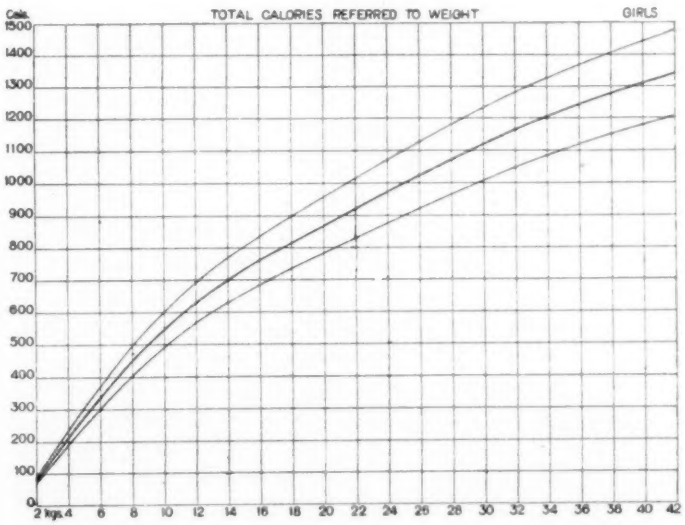

Chart 10.—Basal metabolism of girls, total calories in twenty-four hours at different weights. The curve is projected from 32 kg. upward.

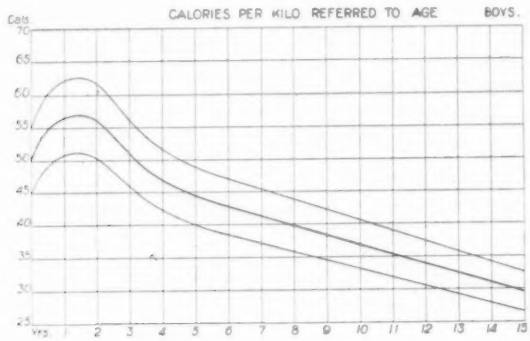

Chart 11.—Basal metabolism, calories per kilogram of body weight for twenty-four hours of boys at different ages. The curve is projected from 12 years upward.

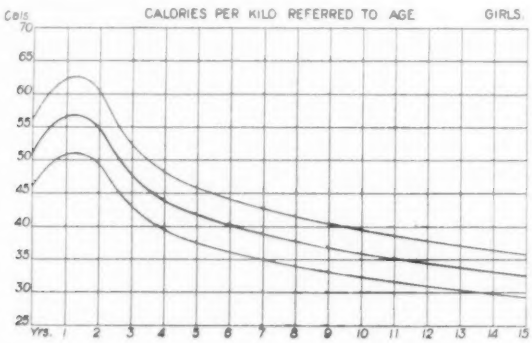

Chart 12.—Basal metabolism, calories per kilogram of body weight for twenty-four hours of girls at different ages. The curve is projected from 12 years upward.

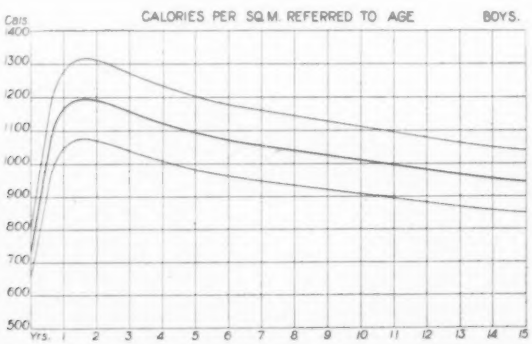

Chart 13.—Basal metabolism, calories per square meter of body surface for twenty-four hours of boys at different ages. The curve is projected from 12 years upward.

ing normal children, however, varies within wide limits, and, consequently, variations occur in their basal metabolism. Variations within 10 per cent. either side of the average curve have been taken arbitrarily as the extreme limits of variation of children with approximately average development. A careful study of the cases outside the 10 per cent. limits has shown that they belong to certain groups.

Children who are above the average height and weight for the age and who have a normal amount of muscle and fat, are found to have a total metabolism of more than 10 per cent. above the average for the age, but within the 10 per cent. variation when applied to the total metabolism for their weight and per unit of body weight and body surface. On the other hand, a child that is of normal development but small for the age, has a total metabolism of more than 10 per cent. below the metabolism for the age but within 10 per cent. of

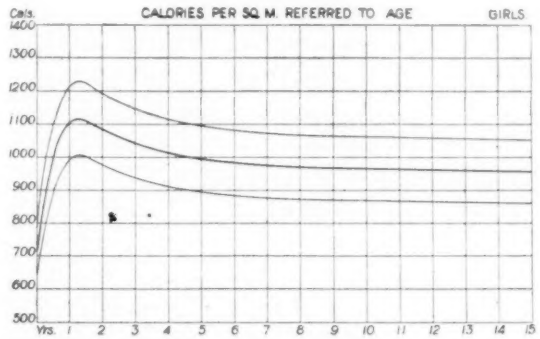

Chart 14.—Basal metabolism, calories per square meter of body surface for twenty-four hours of girls at different ages. The curve is projected from 12 years upward.

the total metabolism for the weight and per unit of body surface and body weight. In both these instances, the total metabolism conforms with the expected metabolism for the weight but not for the age.

Secondly, children who are of average height but over-weight have a total metabolism which usually falls within the average limits for the age and per unit of body surface. Those who are fatter than the average have a metabolism per kilogram of body weight below the 10 per cent. variation, and those who are thinner than the average are more than 10 per cent. above the average; the extent of the variation depending on the relative amount of body fat. This is, in all probability, due to the fact that fat is an inert substance in so far as the energy metabolism is concerned.

Thirdly, in children that are both large and fat, the total metabolism tends to fall within the average limits for the age, but the metabolism per unit of weight and per unit of body surface falls below the 10

per cent. variation; while those children who are small and thin fall above the 10 per cent. variation per unit of weight and per unit of body surface. The metabolism per unit of body surface is less affected in these comparisons than the metabolism per unit of body weight.

These variations in the metabolism are more pronounced in infancy than in later life. Studies of the metabolism of the first two years of life, therefore, are much more difficult to interpret than are those of older children.

These charts are presented as standards of the average basal metabolism of average normal children. The basal metabolism of the majority of cases studied up to date fall within 10 per cent deviation of the average line given in the charts. Physical development of the child plays a part in influencing the metabolism in childhood, but the proportion of body fat plays a still greater part in causing a deviation from the average.

THE REFLEXES IN EARLY INFANCY*

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Gottfried Engstler [1] found that in 232 children in the first week of life, and with sound nervous systems, the plantar reflex consisted of dorsal flexion of the toes, whereas in two children there was plantar flexion and in eight no response was elicited on stroking the sole. Between the second and eighth week, ninety-nine showed dorsal flexion, one child showed plantar flexion, and eight gave no response. In the third half year, sixty presented dorsal flexion, thirty gave no response, and forty had plantar flexion. On the other hand, in the third year, five presented dorsal flexion, five gave no response, and ninety-five had plantar flexion.

M. A. Léri [2] in 166 infants found extension of the toes on stroking the sole the rule at birth and flexion the exception, whereas after three years, flexion was the rule and extension the exception.

John Lovett Morse [3] concluded after the examination of 254 infants during the first twenty-three months of life that

> It is evident that there is no constant plantar reflex in the first year, and that while the reflex approaches the adult reflex during the second year, it is still inconstant. It is also evident that since there is no constant reflex under normal conditions during the first two years, no conclusion can be drawn from the presence, absence, or character of the reflex in the diagnosis of abnormal conditions of the nervous system at this age.

Kalischer [4] as quoted by Morse,[3] states that no conclusions can be drawn concerning the plantar reflex in babies because of the many voluntary motions. If, however, a certain movement constantly follows every irritation and is confined to one side it is significant of disease.

J. P. Crozer Griffith [5] states:

> Some of the reflex movements which persist throughout life, as the plantar and the patellar reflexes, are, on the whole, not so uniformly well shown in the first and second years as later. Others, such as the abdominal, cremasteric, corneal and pupillary reflexes, are fully developed from birth.

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* Received for publication, Jan. 26, 1921.
* Read at the meeting of the Philadelphia Pediatric Society, Jan. 11, 1921.

1. Engstler, G.: Wien. klin. Wchnschr. **18**:567 (June 1) 1905.
2. Léri, M. A.: Gaz. d. mal. infant. **5**:277, 1903.
3. Morse, J. L.: Pediatrics **11**:13 (Jan. 1) 1901.
4. Kalischer: Virchows Arch. f. path. Anat. **155**:486, 1899.
5. Griffith, J. P. C.: The Diseases of Infants and Children, Philadelphia and London, W. B. Saunders Co. **1**:65, 1919.

G. L. Walton and W. E. Paul [6] "question the existence of a characteristic toe reflex at this period" (early Infancy). They note that several times when a baby was examined a second time after a very short interval, the examiner not knowing the little trick being played, the two descriptions of the reflex movements were contradictory.

There are practical difficulties in testing the reflexes in babies not present in older people. The new-born and very young are in a state of almost constant movement, except during sleep, on account of the numberless stimuli from within and without, stimuli to which older children pay no attention. Further, if the leg is held by the examiner while trying to get, say, the plantar jerk, the infant often immediately and persistently resists (holds the leg in rigid flexion or extension) and hence the reflex does not appear. The patellar tendon is very small in babies, and to percuss it is not always easy. In a crying baby often no reflex movement can be obtained on account of the general muscular rigidity, while in the same baby at rest all reflexes may be prompt.

Curiously enough, muscular contraction in other parts of the body (the leg remaining relaxed) did not (in the children I examined) reinforce the knee jerk; e. g., if a child grasped something vigorously in its hand while the knee jerk was being taken, the response was neither quicker nor more rapid, nor did the foot go through a larger arc than when the child was at absolute rest.

I have examined at the Children's Hospital and the Philadelphia General Hospital (Blockley) sixty-nine infants. The ages varied as follows: 1 hour or under, 3; between 1 hour and 24 hours, 11; more than from one day to 7 days, 28; from 8 to 30 days, 15; from 31 to 90 days, 12.

Twenty-seven infants were of negro blood of varying degree and a study of them revealed no racial peculiarity in the reflexes. The children were all free from any disease of the nervous system, save one child (9 days old) who had a congenital peripheral facial palsy.

As to diseases other than a nervous affection, two had been operated on for pyloric stenosis; one had infantile atrophy; one was a premature birth (7 months). In the seven months baby, when the child was 46 days old, the knee jerk was normal; the abdominal jerk was present only on stimulating the lowest third of the belly of the muscle ; the chin jerk was present, and while on the right the plantar jerk consisted in extension of the toes, on the left, during repeated trials, there was no toe movement but only withdrawal of the whole foot. Barring this dif-

6. Walton, G. L., and Paul, W. E.: Nerv. & Ment. Dis. **27**:305 (June) 1900.

ference in the plantar jerk on the two sides, there was no greater variability in the reflexes than is found in healthy infants.

As to the health of the mothers, two had had puerperal eclampsia. The child of one (1 hour old) gave slow, deliberate extension of all toes, a very prompt Achilles jerk, prompt knee jerk on both sides with contralateral adduction of the thigh, and absent scrotal jerk. The child of the other, when 6 days old, gave slow extension of the great toes with no movement of the other toes and normal knee jerks, and percussion of the chin caused twitching of the upper lip. There was one imbecile mother. Her child (26 hours) had normal knee and Achilles jerks and extension of all toes.

The knee jerk was examined in sixty-six children and was present in sixty-one. The ages of the five in whom it was absent were: one less than 1 day; one, 2 days; two, 3 days; one 4 days. It was present in three children less than one hour old. Out of ten more than one hour and less than one day old it was present in nine and absent in one. In those more than one day and less than 8 days old, it was present in twenty-one and absent in four. It was present in all children more than 8 days old. In one white boy, 6 days old, however, who showed no signs of disease of any kind, the right knee jerk was prompt and easily gotten; the left was absent. He also had no true plantar jerk but only withdrawal of the foot; no cremasteric jerk and no Achilles jerk. His case is proof of the fact that differences in the knee jerk on the two sides in a child is not indicative of disease. In one girl in whom the knee jerk was absent at 19 hours, it was present ten days later, and in a boy in whom it was absent at 3 days it was present four days later.

Even in the adult the Achilles jerk is absent sometimes, and its presence or absence can only be determined after careful examination in a proper posture. I examined fifty-two of the infants and found it absent in thirty. In two examined within one hour after birth it was present in one, absent in one. Whereas, in many of the children the knee jerk was as good as in the adult, in most of the children the Achilles jerk was only obtained after many attempts and then it usually was very feeble; occasionally, it was as good as one ever sees in adults.

There are many difficulties in studying the plantar jerk in infants on account of their restlessness. I regarded as a plantar reflex only flexion or extension of the toes. In nine instances, though the toes moved, the movement was so erratic that I could form no judgment. In nineteen others there was no movement of the toes of any kind, no matter what part of the sole was stimulated, and, incidentally, I found

that stroking the outer edge of the sole, in general, gave the best response. In twenty-five cases there was definite extension of the toes, and in sixteen there was flexion. Within an hour of birth the plantar jerk may be well established. Thus, one boy, one hour old, showed beautiful and deliberate extension of all the toes with fanning. His knee and Achilles jerks were also well developed. Another boy of the same age showed a constant slow flexion and a third had no plantar response of any kind. In one boy, 2 days old, and in another 3 days old, there was a quick extension of the toes followed by slow flexion and bunching of them together as if trying to grasp an object with all the toes at once. The grasping movement was just that which our far off arboreal ancestors must have made as they swung from bough to bough and caught at support with the feet. In another boy, 4 days old there was first extension and then flexion of the great toe without any movement in the other toes.

I did not examine the chin jerk in a large enough number (twenty-five) for my results to have any statistical value. Suffice to say, it may be but is not always present within one day of birth. In one girl, 31 days old, the reflex consisted in a closing of the lips, in another girl, 8 days old, and a boy, 6 days old, there was only a twitching of the upper lip.

I examined the abdominal jerk in twenty infants and found it absent in seven. It was present in two under 4 days old; on the other hand, it was absent in four out of seven between 2 and 3 months old. In four it was obtained only on stroking the skin over the lowest third of the rectus abdominis muscle.

CONCLUSIONS

1. The deep and superficial reflexes (i. e., knee, Achilles, chin, plantar, abdominal) may be present at birth but the absence of one or all in early infancy does not indicate disease.

2. The plantar jerk is very variable. It may be absent up to the third month or longer, or there may be extension or flexion of the toes, or at one time there may be one movement, at another the other. Most frequently there is extension. The movement may be rapid or deliberate.

3. The Achilles jerk is very frequently absent at birth. (How late in life it may appear is unknown).

4. Sometimes the abdominal jerk can be obtained only on stimulating the lowest third of the muscle.

5. Whether the occasional absence of the knee jerk in healthy adults (1 in 500) is congenital or the result of disease in early life cannot

be decided in the absence of a complete medical history. (Diphtheria may permanently abolish it without persistence of other signs of nervous involvment).

6. When the reflexes appear after birth, their appearance does not occur in any regular order.

THE FACTOR OF THE POSITION OF THE DIAPHRAGM IN ROENTGEN-RAY DIAGNOSIS OF ENLARGED THYMUS *

H. J. GERSTENBERGER
CLEVELAND

The diagnosis of the presence of an enlarged thymus by percussion, I believe, has been considered by most physicians to be a difficult and uncertain performance.. Therefore, the advent of roentgen ray was hailed and is generally still believed to be a safe and dependable diagnostic method as applied to the thymus.

An experience during the past winter has convinced me that the diagnosis of an enlarged thymus with the aid of the roentgen ray is also liable frequently to be very uncertain and to be a source of erroneous interpretations and findings.

A brief recapitulation of this experience will bring out the interesting points.

REPORT OF CASE

In July, 1919, in the absence of the family physician in the army, I was called to atttend the child of a colleague, whose skin was scaly and itchy and who was breathing with difficulty. The boy, 12 months old, was quite well developed and nourished, showed a mild rickets, a distinct but slight eczema of the face, chest and arms, causing a great deal of itching, and a decided emphysema, accompanied by wheezing and a somewhat labored expiration. The spleen was not palpable and the posterior cervical lymph nodes were somewhat increased in size and in number, as is usually the case in exudative children.

The child had since his third week received a Walker-Gordon mixture, and recently has been given straight cow's milk and vegetables.

The condition was considered to be due either to a hypersensitiveness to some food constituent or to an exudative diathesis.

A skimmed milk diet, atropin sulphate in increasing drop doses, to physiologic limit, and 10 per cent. tricalcium phosphate in cod liver oil were ordered, and were followed by a decided improvement.

During August, 1919, the family physician returned, found an increased thymic dulness, and requested a roentgenogram, which showed a wide shadow, especially to the right but also to the left of the sternum, and which the roentgenologist stated was caused by an enlarged thymus (Fig. 1). Consequently the family physician stopped the atropin and had the thymus exposed every three to four weeks for a total of five exposures.

The condition of the child, however, grew slightly worse instead of better, and the thymic shadow remained the same, so that the roentgenologist advised against further exposures. The family physician then reordered the atropin in the form of belladonna in small doses. The child improved again and breathed in a nearly normal manner during the latter half of November, 1919.

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* From the Departments of Pediatrics of Lakeside Hospital and of Western Reserve Medical School.

During the first days of December he contracted a cold and in the night of December 3 he became severely asthmatic and somewhat cyanotic. I saw him with the family physician December 4 and he was then taken to Lakeside Hospital for diagnosis and treatment.

Nov. 10, 1919, the family physician had his assistant make cutaneous scratch tests with different proteins in simple saline solution, which were all negative. The proteins used were as follows: wheat, cow's milk, rice, potatoes, egg white, beef, oats, tomato, horse hair, pneumococci, staphylococci and ragweed.

December 4 at the hospital the diet and therapy of July, 1919, were reordered and the child improved.

December 5 a roentgenogram was taken, which showed a much smaller shadow than had ever been seen in this case before and really one that was practically normal (Fig. 2). This finding convinced the family physician that after all the improvement noticed in the latter half of November, 1919, was due to a reduction in the size of the thymus, which had occurred since the last picture had been taken in October.

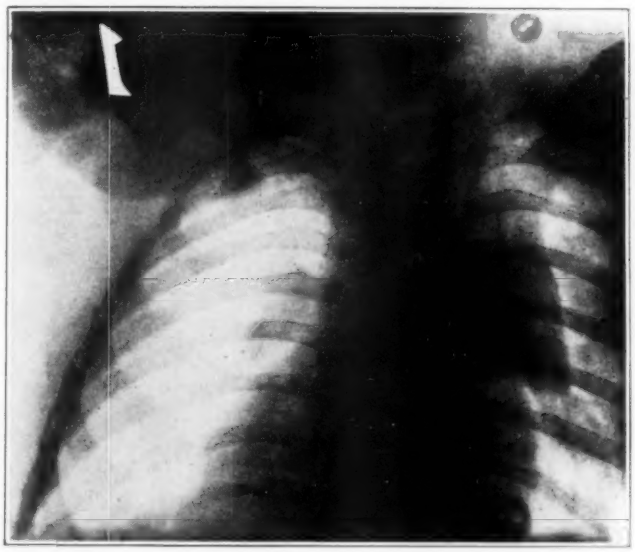

Fig. 1.—Roentgenogram made Aug. 5, 1919, showing decided shadow in thymic region, especially on right side. Note sharp, angular projection at right lower end of shadow.

Intracutaneous sensitization tests were carried out at the hospital from December 8 to 13 and showed the child to be negative to cow's milk casein, cow's milk albumin, strawberry, orange and tomato, questionably positive to cow's milk allergen and enormously sensitive to egg yolk allergen and egg yolk albumin. The latter two tests were accompanied by a severe anaphylactic shock that was relieved by the administration of adrenalin subcutaneously.

December 16 two roentgenograms were taken, both of which showed an abnormally large shadow in the thymic region, in other words, just the opposite from the last findings in the plates of December 5.

The improvement of the child, that was still getting atropin in increasing doses, cod liver oil with tri-calcium phosphate and skimmed milk, continued, and did not correspond with the decided reversal in the shadow in the roentgenogram.

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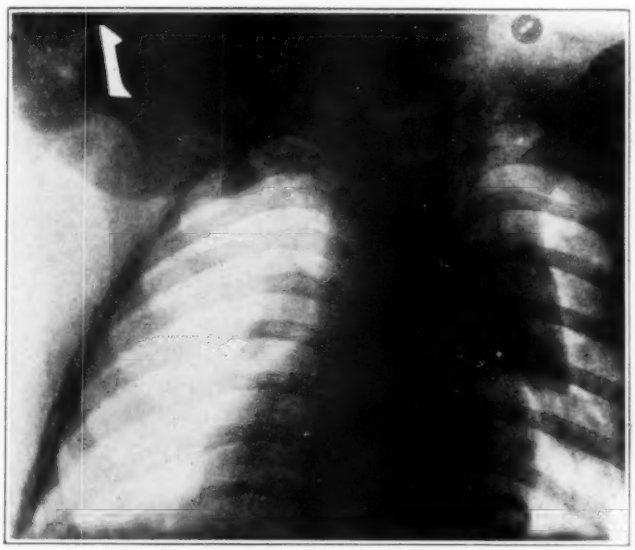

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A fluoroscopic examination was immediately made and succeeded in establishing the interesting fact that the two extremes of normal narrow shadow, on the one hand, and a markedly wide and seemingly abnormal thymic shadow on the other, corresponded in the former with the end of a deep inspiration and in the latter with the end of a deep and forceful expiration.

The development of the shadow in its most marked form under the fluoroscope may well be compared with the filling of miniature lungs which paradoxically decrease in size with inspiration and collapse entirely with a deep taking in of air. In order, however, to get this markedly increased shadow which in this patient was always greater on the right than on the left and frequently present only on the right side, especially if the expiration had not been of the extreme type, it was necessary to have the child cry vigorously and establish a prolonged and forceful expiration.

It is especially significant to note that the expiration had to be forceful, for at times I saw a prolonged expiration produce an increase in the shadow of the thymic area without presenting the beautifully clear picture to either side of the vertebral column, simulating as stated above the filling to capacity of a miniature pair of lungs with distinct outlines, definite and sharp borders. Whenever I have seen this picture produced under the fluoroscope it has seemed to me that the diaphragm moved distinctly upward and was directly or indirectly

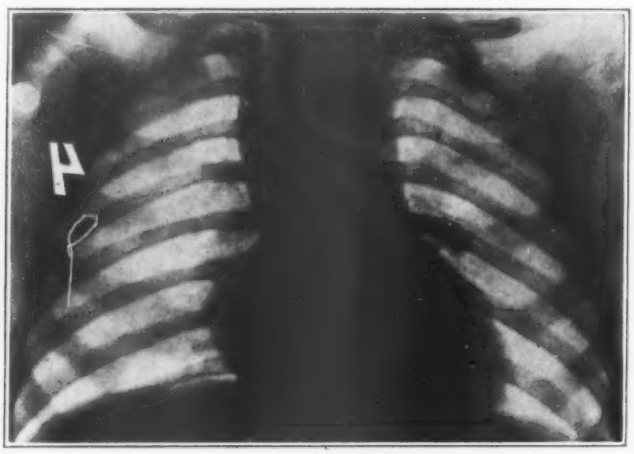

Fig. 2.—Roentgenogram made Dec. 5, 1919, after repeated exposures (from four to five). Shadow in thymic region decidedly less and practically normal.

responsible for it. Likewise, I have always seen a distinct descent of the diaphragm, accompanied by a very deep and very voluminous taking in of air, precede the rapid and complete disappearance of the abnormal shadow.

Unless this deep inspiration and distinct drop in the position of the diaphragm occurred, the shadow would only get smaller and indistinct as it partially receded toward the middle line.

Whenever a clear and marked shadow was seen under the fluoroscope one could note the outlines of the lobelike shadow on either side, but again always more distinctly on the right than on the left, and a definite and sharp but slightly rounded angle at the extreme outer and lower pole, separated by a clear rather straight space from the heart and distinctly away from it and pointing out into the area of the lungs.

At no time was any pulsation noticed in any of the shadows or in any part of a shadow, when seen under the fluoroscope in this patient.

It is further significant that only when a forceful expiration was established could a lobelike shadow be seen to the left of the middle line and then never as clearly as on the right side. When the shadow on the right side was medium in degree no definitely outlined form could be seen on the left side. All of these pictures were seen much better in the fluoroscope than in the roentgenogram. However, on three of the pictures a less intensive, lobelike somewhat hazy shadow can be seen to the left of the middle line, which together with the shadow on the right side sits over the heart in the form of a blunted cone with a distinct lower border *separated at the side,* especially on the right side of the heart, *by a clear sharp space as described above, and forming a distinct angle, pointing outward and away from the heart.*

The child went home for the Christmas holidays and returned to the hospital January 7, where it remained for ten days. During this time four attempts were made to register on plates in prompt and, if possible, in immediate succession pictures of the two extremes seen with the fluoroscope, so as to be able to present to those interested in the roentgen-ray diagnosis and therapy of the thymic gland, the customary before treatment and after treatment pictures only obtained, however, in this child both on the same day and at the same sitting.

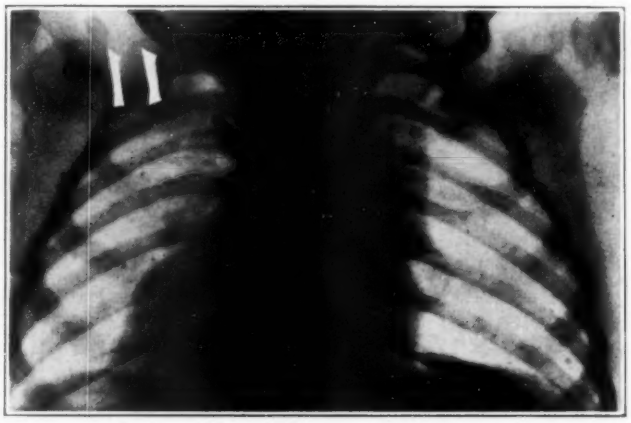

Fig. 3.—Roentgenogram made Jan. 17, 1920, showing return of decided shadow in thymic region, especially on right side, and practically identical with shadow shown in Figure 1. Note again the presence of the same sharp, angular projection at the right lower end of shadow.

January 8 both plates showed wide shadows to the left as well as to the right, but much more distinctly on the latter side.

January 12 it was again impossible to obtain photographs during the height of inspiration and both plates showed in consequence decided shadows to the right.

The pictures taken January 15 were out of focus and could not be utilized.

Finally January 17 we were able to get first a markedly wide shadow involving both sides (Fig. 3) and immediately afterward a narrow shadow that was practically normal (Fig. 4) and just like the two obtained December 5. In other words, we did obtain in the same child at the same sitting both a normal and an abnormal shadow in the thymic region, certainly a difficult finding to interpret.

COMMENT

Numerous questions difficult to answer immediately arise. Is this shadow due to the thymic gland or to something else as the superior vena cava or the right auricle? Is the thymus really abnormally large or is its size actually within the variable normal limits, and is it brought into the plate in the form of an abnormal shadow simply as a result of the operation of some abnormal factor such as an abnormally increased intrathoracic pressure especially in this child, whose lungs, owing to a possibly at times still existing construction of the bronchioles, might retain more air than those of a normal child at a given stage of respiration and so might enable the production of an intrathoracic pressure in forced expiration that would be greater than that found in a normal child?

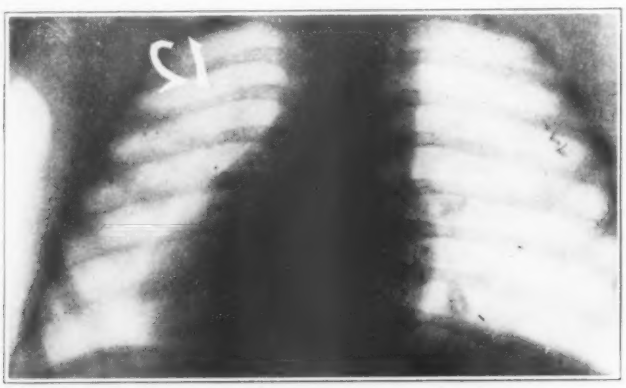

Fig. 4.—Roentgenogram made Jan. 17, 1920, five minutes after obtaining Figure 3, and showing just the opposite finding, namely, a practically normal shadow in the thymic region, and corresponding to Figure 2 obtained Dec. 5, 1919.

Or are the intrathoracic factors causing the thymus to stand out in this so-called abnormal manner really normal and simply abnormally utilized in the sense of hypereffectiveness?

For instance, is it possible that one normal child can push his diaphragm up more effectively or increase his intrathoracic pressure more decidedly than another normal child of the same age and weight? Again, if it is the thymus are its lobes mechanically pushed upward and outward by the ascent of the diaphragm or are they enlarged by a sudden filling with blood as a result of the establishment of an abnormal disproportion between the outflow and the inflow of blood into the gland? Or is its coming out into view due only slightly to the possible effect of the vascular supply on the thymus and mainly due to a following of a mechanical path of least resistance, as a result of a markedly increased intrathoracic pressure produced by various factors, but

recorded best by the ascent of the diaphragm? Does the varying topographical relation of the lungs to the thymus play a part?

Why is the shadow more often present on the right side, and when also present on the left, less distinct than on the right? Is this shadow on the right side really due to the thymus or is it produced by the superior vena cava and right auricle? If it is produced by the thymus is it caused by its close proximity to the just mentioned venous structures? Or is it due to possibly more rapid enlargement of the right lobe than of the left lobe of the thymus, or is it mechanically advantageous under an increased intrathoracic pressure for the right lobe to move out before the left?

Is such a freedom of mechanical mobility of the lobes within the sac of the thymus possible, or is it necessary that a force from within such as would be represented by the filling of the thymus with blood be developed to force the lobe outward in the manner observed?

These questions are all of importance, and I do not see how we can answer them with positiveness until further data are at hand. Definite contradictions exist in the literature regarding most of these points. I have been unable to find satisfactory data regarding the blood supply of the thymus, especially regarding the proportion existing between the inflow and outflow.

Rehn,[1] Hotz [2] and Hart all think that the exit venous channels are relatively better developed than arterial inflow vessels, and yet Klose,[3] notwithstanding the fact that he quotes Hotz and Hart as his authorities, expresses just the opposite view, based mainly on the findings of Siegel,[4] who reported in one case the presence of a net work of fine veins on the circumference of the gland and the absence of larger venous channels. Siegel, however, does not attach the same generalization and significance to this individual observation.

2. Gesellsch. f. Chir., April, 1906, 35th Congress, p. 364.

Rehn, in 1906, observed at operations on the thymus a regular downward movement of the thymus with inspiration and an ascent with expiration. He makes the further comment that the more forceful the inspiration or expiration the greater the descent of the thymus in the one instance and the ascent in the other, and with deep inspiration the thymic gland is aspirated as it were into the thorax, whereas by severe expiration as during crying, coughing, the gland is forced up out of the chest.

Rehn claims that this physiologic up and down movement of the thymus is responsible, when it is enlarged, for the difficulty in breathing

1. Rehn, L.: Die Thymusstenose under der Thymustod, Verhandl. d. Deutsch.
2. Hotz, G.: Die Ursachen des Thymustodes, Beitr. z. klin. Chir. **55**:515, 1907.
3. Klose, H.: Beiträge zur Pathologie und Klinik der Thymusdrüse, Jahrb. f. Kinderh. **78**:653, 1913.
4. Siegel: Berl. klin. Wchnschr., No. 40, 1896.

that is noticed in some cases at beginning of inspiration, by fitting better and more snugly in the upper opening of the chest at the end of expiration and so acting as a more tightly fitting valve.

Benjamin and Goett,[5] in presenting their experiences at Pfaundler's clinic with roentgen-ray studies of the thymic gland in infants, report an exactly similar observation as the one made by me in this patient, with this difference, however, that the shadow, as observed under the fluoroscope, increased with the descent of the diaphragm and decreased with the ascent. They come to the provisional conclusion that the abnormal picture was produced by the distention of the vena cava and not by the thymus. Later on in their article, however, they modify this conclusion by stating that, according to their recollection, the child cried at the time when the picture of the abnormal shadow was obtained, and was quiet when the good shadow was procured.

If their provisional conclusion is the right one, then the interpretation given by them would seem to be correct from a physiologic point of view. But, as they remark, there is no uniformity of opinion in the literature as to whether the heart in adults during inspiration is wider or smaller than it is during expiration, or vice versa.

Grödel [6] and Dietlen [7] are of the opinion that the descent of the diaphragm produces an inspiratory narrowing of the cardiac shadow, and that the ascent produces an expiratory widening, while Holzknecht and Hafbauer (quoted by Benjamin and Goett) find a widening of the shadow during the entire inspiration.

Dietlen bridges the two contradictory views by admitting a widening of the cardiac shadow in early inspiration due to the sudden filling of the superior vena cava and right auricle as the result of the suction effect during the early part of inspiration.

Benjamin and Goett also quote d'Oelsnitz and Paschetta [8] as having called attention to the part played in infants by the large vessels in the widening of the shadow at the base of the heart and to the fact that this widening is influenced by respiration in the sense that the shadow becomes wider in expiration and narrower in inspiration.

In addition these authors further claim that an enlarged thymus appears mainly to the left of the vertebral column. So one can readily agree with Benjamin and Goett that a subject that at first glance seems

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5. Benjamin, E., and Goett, T.: Zur Deutung des Thoraxradiogramms beim Säugling, Deutsch. Arch. f. klin. Med. **107**:509, 1912.

6. Grödel, F. M.: Röntgenkinematographische Studien über Herzgrösse und Herzlage, Ztschr. f. klin. Med. **72**:310, 1911.

7. Dietlen, H.: Ergebnisse des medizinischen Röntgenverfahrens für die Physiologie, Ergebn. d. Physiol. **10**: 1910.

8. d'Oelsnitz and Paschetta: Bull. de la Soc. de Pediat, Paris, 457, 1911. Quoted by Benjamin and Goett: Deutsch. Arch. f. klin. Med. **107**:514, 516, 1912.

simple and clear, when viewed from the standpoint of opinions as written down in the literature, is complicated and difficult.

Benjamin and Goett were checking Hochsinger's [9] claim that the stridor congenitus was caused by the presence of an enlarged thymus, and that this enlarged thymus could be found in the roentgenogram. They had occasion to perform a necropsy on a 7-month-old infant killed in an accident and who also had a congenital stridor and of whose thymic area a roentgenogram had been made. A 20 gm. thymus was found and neither was it nor any other gland in the place where a shadow had been obtained in the plate. This fact, of course, does not prove that the thymus was not concerned in producing the shadow during life.

Again in another infant, the child of a wet nurse, who had never had a congenital stridor but who had frequently showed in the roentgenogram the presence of Hochsinger's shadow and on whom a necropsy was made after an acute illness of six days' duration they found only a very small thymus weighing hardly 3 gm., and concluded that it could not have been a factor in the production of the shadow in this child.

It is well to remember in this connection that Benjamin and Goett found their shadow, as observed under the fluoroscope, during inspiration and not during expiration, and therefore they could hardly expect the thymus to be a factor unless it be of enormous size.

Benjamin and Lange [10] state "that the breadth of the upper mediastinal shadow varies normally with the age and general condition of the patient, and since it may vary from time to time in the same patient the roentgen-ray diagnosis is not always easy or free from error." They continue further, "in one case not included in this series an apparently normal shadow was obtained when the child was quiet or sleeping, but during a restless crying spell the thymic shadow became greatly enlarged."

"The roentgen-ray shadows of congenital heart enlargements are often confused with thymic enlargements. In all cases in which the roentgen-ray diagnosis seemed doubtful, a roentgen-ray exposure was given as a therapeutic test and this test has proven very reliable."

In other words, Benjamin and Lange have made an observation similar to mine by getting a wide shadow in a crying child—picture in all probability taken in decided expiration—and a normal shadow while the child was quiet and sleeping—picture probably taken in inspiration or in very superficial expiration.

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9. Hochsinger: Stridor thymic infant, Wien. med. Wchnschr., No. 45, 1903; No. 33, 1910.

10. Benjamin, J. E., and Lange, S.: Report of Nineteen Cases of Hyperphasia of the Thymus Gland, Treated by the Roentgen Rays, Arch. Pediat. **35**:71, 1918.

These authors do not, however, indicate how they decided in this case, by therapeutic tests or otherwise, whether the infant had an abnormal thymus or not as this child was not a part of the series which they were reporting. Nor do they go into the possible rôle that respiration or the diaphragm might play in the production of this shadow picture.

It would seem as if the therapeutic roentgen-ray tests based on finding a normal shadow would be valueless unless checked by observations made at two different respiratory phases and especially with the aid of the fluoroscope.

The patient forming the basis of this contribution is an excellent example of the case in point. Here we have a boy who has a history of difficult breathing, especially during expiration, accompanied by slight cyanosis, symptoms which very well fit into the picture of a case of so-called thymic asthma, just as they also may be just as well a part of the clinical picture of a child sensitive to proteins, or of an exudative child with asthma. Although he has received sufficient exposures to the roentgen ray, this boy can show, just as you wish, either a normal or an abnormal shadow of the thymic region, corresponding with marked inspiration in the former, and with deep and forced expiration in the latter, but not running parallel to his general condition.

This patient has a definitely abnormal shadow that, in all probability, is produced by forcing the thymus upward and outward, and yet it is also most probable that this thymus is not mechanically concerned in giving him his respiratory difficulty. And in the face of a marked protein hypersensitiveness, accompanied by a mild eczema and expiratory asthma, it is impossible to claim that this boy is one of the thymic cases that are refractory to roentgen-ray therapy and that for this reason his thymus gland has not decreased in size. In other words, unless we find in this boy what, at the present time is considered to be a normal shadow, both in expiration as well as in inspiration, under the fluoroscope, as well as in the roentgen-ray plate, we will not be able to give credit for his improvement to the effect of the roentgen ray on the thymus gland.

Of course seemingly definite cases of clinical thymic asthma in the newly-born have been rapidly and definitely relieved of their symptoms by exposure to the roentgen ray. Usually their improvement has been accompanied by decided narrowing of the shadows, so that the conclusion has been drawn that the betterment was due to the reduction in size of the thymic gland. But in the light of the present case under discussion we cannot be sure that the roentgen ray actually reduced the size of the gland and still it really may have accomplished just that. Further observation on the basis of the experience attained with this patient will help to settle that question. It may be that as a result of the exposure to the roentgen ray the child's condition was improved,

and as a result he was more quiet when later pictures were taken, so that normal shadow may have been produced because the child was more comfortable and did not cry and squirm when the pictures were being taken, rather than because the thymic gland had been decreased in size. It is conceivable that the roentgen ray might influence an internal secretion of the thymus and as a result remove the agent that is responsible for the asthma without at the same time causing any change in the actual size of the gland.

It is necessary to call attention to one of the arguments used by Benjamin and Goett in favor of their opinion that the abnormally wide shadow found in normal children varies according to their condition; the well nourished showing a wide shadow, the poorly nourished a normal shadow. They attributed the variations to the difference in blood volume between the two types of children. While this difference does exist, it is equally true that the size of the thymus gland seems to follow the same rule, being small in the skinny child and large in the well nourished, so that this argument can be applied to one theory with no greater justification than it can to the other. However, one can see how in the well nourished child both factors could operate together to the advantage of a wide shadow, whether it be thymic or nonthymic.

SUMMARY

1. The case of a patient, 17 months old, is presented who under the fluoroscope and in the roentgenogram shows a wide shadow in the thymic region which, according to present conception, is considered abnormal.

2. This shadow in its most marked form extends both to the right and to the left of the vertebral column, while in its mediumly severe form it projects out only to the right.

3. This wide shadow is sharp in outline and has a definite shape, similar to that of a miniature lung, *with an angle at the lower, outer, right side which seemingly cannot be produced by the vena cava, nor by the auricle, as it extends clearly separated from the heart out into the lung area,* and not toward the middle line, as would be the case if a distended auricle were responsible for it. No pulsations were ever seen in the shadow or any part of it, and until contradictory evidence is advanced the shadow is accepted as of thymic origin.

4. *The abnormal shadow can be replaced by one of normal size if the patient takes a deep inspiration.*

5. *The abnormal shadow can be brought out in its most marked form by having the patient expire deeply and forcefully. Both observations can clearly be seen with the aid of a fluoroscope.*

6. The diaphragm, as observed under the fluoroscope, rises with the development of the abnormal shadow and descends with the development of the normal.

7. Frequent exposures to the roentgen ray for a period of over five months have not changed the size of the thymus, nor have they influenced in a dependable manner the general condition of the patient. As a matter of fact, the patient has improved, while his thymic shadow has remained the same. The general improvement can well be explained by the dietetic changes instituted and the administration of atropin.

8. In producing the abnormal shadow in expiration the diaphragm seems to be the most important individual factor, either directly and mechanically by pushing the heart upward and so forcing the thymus in the same direction and also outward when ascending markedly, or indirectly by increasing the intrathoracic pressure to a sufficient degree to move the thymus upward in a mechanical manner, or by retarding decidedly and in forced extreme expiration nearly entirely the venous outflow from the thymus, while still permitting its arterial inflow, and by so creating an acute engorgement and enlargement of the gland, which would cause the above factors to operate even more advantageously, and which would explain better than anything else the miniature lung-like development of the shadow under the fluoroscope.

9. In establishing the return to a normal picture the descent of the diaphragm would seem to operate by relieving the causative factors indicated above, and so allowing the thymus to descend down toward and over the middle of the heart and to empty itself rapidly following the suction effect of inspiration, especially when deep.

10. The wide shadows in their most marked form appeared on both sides, especially when viewed under the fluoroscope, but more on the right than on the left. In mediumly wide shadows only the right side was represented. Consequently, it is not justifiable to conclude that a picture showing a shadow limited to the right side comes from a patient who is unable to develop a shadow on the left side as well, if given the proper conditions, nor can we conclude on the same basis that this shadow on the right side has nothing to do with the thymus gland and has been produced by some other factor such as an enlargement of the vena cava or auricle.

CONCLUSIONS

1. In order to have accurate knowledge as to the actual size of the thymus it is necessary to examine the gland under the fluoroscope and watch its position and condition at the various phases of respiration, especially in the development and at the end of a somewhat

prolonged and forceful expiration, and again during a deep inspiration following the type of expiration just described.

2. If roentgenograms are made, an attempt should be made to get pictures both in deep inspiration and deep expiration. It may be well likewise to first attempt to get a picture while the child is breathing quietly and normally. It must, however, be realized that to procure roentgenograms of the thymus in the different respiratory phases is most difficult, uncertain and time consuming, and, therefore, it is suggested that the fluoroscope be always used to check the findings of the roentgenogram.

3. That unless the thymic shadow is observed as suggested above, a normal shadow obtained following a previous abnormal one does not mean an actual reduction in size of the gland.

4. The findings reported might explain the contradiction of reports according to which clinical improvement was obtained by exposure to the roentgen ray in cases where the thymic shadow was normal. It is possible that these cases really had an enlarged thymus, and that the picture just happened to have been taken during deep inspiration or very superficial expiration. It is, of course, also possible that in such cases the enlargement of the thymus is in the anteroposterior direction.

5. In all probability the position of the diaphragm directly and mechanically, or indirectly by influencing the intrathoracic pressure is more responsible than any other one factor for the difference observed in the pictures taken on the one hand in extreme inspiration and on the other at the end of a marked and forceful expiration.[11]

6. So called improvement in thymic shadow, as presented in Figures 2 and 4, was demonstrated by the fluoroscope to be due not to a reduction in the size of the thymus, but to a change of local conditions in this region, causing, at the end of forced expiration a wide, seemingly abnormal shadow, and at the end of deep inspiration, a narrow, seemingly normal shadow.

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11. Von Reuss, A.: Die Krankheiten des Neugeborenen, 1914, p. 263; Friedlander, A.: Enlargement of the Thymus Treated by the Roentgen Ray, Tr. Am. Pediat. Soc. **29**:80, 81, 1917.

EMPYEMA IN CHILDREN

WITH ANALYSIS OF ONE-HUNDRED AND SEVENTY-TWO CASES *

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During the last three or four years, there has been a return to closed methods of drainage for empyema, with a rather more widespread adoption of these principles than on any of the previous occasions when this form of treatment had been adopted only to be discarded again partially. It is our belief that the causes of this widespread change or modification in treatment are the occurrence of streptococcus empyema in army camps, with a very high mortality attendant on rib resection, and the very prevalent misconception as to the action of the lungs in the presence of an open thorax. That there is some merit in draining pus from the chest, without leaving the pleural cavity open to the air, is apparent. From a study of the literature and an analysis of 172 of our own cases in which operation was done during what might be called this transitional period we have endeavored to ascertain what merits this form of treatment has, and to what class of cases among children it is applicable.

Every few years a paper is written emphasizing the importance of not allowing air to enter the pleural cavity in draining it and advocating some particular device for accomplishing this end. Thus Robinson devised a very clever scheme of trephining a rib and applying suction apparatus. Von Eberts devised another technic of applying suction with a Politzer bag. Holt and Remsen described still other modifications. All these methods were based on the same general principles.

In more recent years, the same principles have been advocated again with changes in the devices used. Of the more recent methods may be mentioned those of Mazingo, Phillips and Whittemore. Some clinicians advocate irrigating with surgical solution of chlorinated soda. Some apply continuous suction, others intermittent suction, but all lay claim to success partly because they do not allow air to enter the pleural cavity and partly on account of their own particular apparatus.

It is extraordinary that the belief should be so widespread that an opening in the thorax necessarily causes collapse of the lung. It would seem that almost every surgeon who has ever operated for empyema must have observed, after evacuation of the pus, how the lung expands and contracts with respiratory efforts. At times the lung comes directly to the thoracotomy opening. It likewise seems strange that

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many surgeons who perform these operations and have observed the phenomenon of the lung expanding and contracting with respiration at times and not at other times, do not ask themselves why this is so. Apparently they are contented with the statement that the lung on the affected side collapses in the presence of an open thoracotomy wound and does not in the presence of a closed wound.

In connection with these points, the experimental work of Graham and Bell is most interesting and also of great practical importance. They have shown that in the normal chest an open pneumothorax affects both sides almost equally, and that it is not until the structures of the mediastinum are held rigid, as a result of adhesions or inflammatory processes, that marked differences in the two sides of the chest occur. They also show that respiratory difficulties are dependent on the relation between the amount of air entering the thoracotomy wound and that entering the lungs. Theoretically, in a normal chest, the opening would be smaller than 2 inches by $4\frac{1}{10}$ inches to avoid respiratory embarrassment. In cases of empyema of long standing, with adhesions and a firm mediastinum, a much larger opening can be made without respiratory embarrassment ensuing. These factors make deferred operation safer than an operation done too early. This is true of all cases of empyema but particularly of streptococcus cases. It is interesting to note that at the Children's Hospital, five or six years ago, as a result of clinical experience, the same conclusion was reached which Graham and Bell have since clearly demonstrated to be true by experimental work.

Our rule, adopted at that time, was not to operate, except in the presence of frank pus and never in the presence of pneumonia. When we have not adhered to this rule consistently, we have usually regretted breaking it.

The matter of deferred operation in children is often automatically taken care of for the surgeon, through the fact that the diagnosis is considerably more difficult to make in the child than in the adult. As pointed out by Morse and Griffith, the crisis of pneumonia is less clearly defined; the physical signs are less easy to interpret and the roentgen-ray findings are often confusing. The needle must be depended on for a final diagnosis in cases of doubt. As an example of this late diagnosis in children, in our series of 172 cases, the diagnosis was made under two weeks after pneumonia in only seventy-nine cases. In eighty-five cases it was made later than two weeks after the pneumonia. In eight cases the duration was not determined. Of the seventy-nine cases, the diagnosis was made immediately after the crisis of the pneumonia only in those patients who were being treated in the hospital for their pneumonia. In many of the eighty-five cases

diagnosed over two weeks after the crisis of pneumonia, the diagnosis was frequently not made for many weeks or even months afterward. As a general rule, it may be stated that the younger the patient, the more difficult the diagnosis. Among our patients, the youngest was one month old; the eldest 12 years. There were twenty-eight patients under 2 years of age; thirty-four between 2 and 3, and the remainder between 3 and 12 years of age.

In the series there were four cases of bilateral empyema. The right pleural cavity was affected in seventy-three patients and the left in ninety-five.

Several factors enter into the question of mortality in children which cannot be defined so clearly in the adult. The age of the patient is of great significance. Nathan reports seventy-six cases under 2 years with a mortality of 78 per cent. Holt reports 127 cases with a mortality of 65 per cent., with both types of drainage. With closed drainage and siphonage he reports the slightly lower mortality of 60 per cent. In our own series there were twenty-eight cases under 2 years with a mortality of 32 per cent.

Holt reports twenty-seven cases over 2 years of age with a mortality of 16 per cent. Dowd reports 285 cases in children between the ages of 2 and 14 years with a mortality of 25.6 per cent. Our mortality in 144 cases between the ages of 2 and 12, which may be compared with his, is 13.8 per cent. Including our cases under 2 years with the others, 172 in all, the mortality is 16 per cent.

The factor of next importance to age and the condition of the patient is the type of the infection. Holt reports a mortality of 56 per cent. with pneumococcus infection; 79 per cent. with streptococcus and 50 per cent. with staphylcoccus infection.

In our cases, ninety-nine had a pneumomoccus infection, with a 12 per cent. mortality and twenty-six had a streptococcus infection with a mortality of 27 per cent. Four cases in which a pure culture of staphylococcus was obtained were without mortality. The remaining cases were either mixed infections or unknown.

It is obvious that the above facts must be considered in connection with the methods of treatment adopted. We have analyzed our series to determine the results of open or closed drainage as a whole, and also with reference to the age of the patient and the kind of infection. One hundred and fifty-four patients were subjected to rib resection. Three had double empyema. Twenty-six patients died, a mortality of a little over 16 per cent. Two of the three patients with double empyema recovered, both having both sides open at the same time. Of the 154 patients subjected to rib resection, ninety-two had a pneumococcus infection following the pneumonia. Among them twelve

deaths occurred, a mortality of about 13 per cent. Twenty-three had a streptococcus infection. Seven died, a mortality of 30.4 per cent. The four patients with a staphylococcus infection submitted to rib resection, recovered. Eighteen patients were treated by the closed method. Three died, a mortality of 16.6 per cent., a mortality identical with that in the rib resection cases. Of these eighteen patients, seven had a pneumococcus infection, with no deaths; three had a streptococcus infection, with no deaths; these eighteen patients had closed drainage; nine later required rib resection either to provide adequate drainage or to free the lung to allow expansion and obliteration of the cavity. Whereas children do not stand rough handling and hemorrhage as well as adults, the lung can be freed gently, sufficiently to allow it to expand without causing marked bleeding.

Of the 154 rib resections, 21 were done on patients under 2 years of age. Six deaths occurred, a mortality of 29 per cent. One hundred and thirty-three patients were over 2 years of age. Twenty deaths occurred, a mortality of 15 per cent. Seven patients under 2 years of age were operated on by the closed method of drainage with a mortality of 42.8 per cent. Eleven patients more than 2 years of age all survived the operation.

The question of anesthesia has not been considered enough. Ether was given to the majority of our patients. Procain and epinephrin anesthesia was used in fourteen rib resections with only one death. Although it is often impossible to do a rib resection in children under local anesthesia, it could have been used to advantage more frequently and should always be used with intercostal drainage.

The duration of convalescence with closed and open drainage seems to be the same on the average. With the closed drainage, one patient was well in a week with the wound practically healed but in the majority of cases four to five weeks elapsed as in the rib resection cases.

From this study of 172 cases, the following conclusions seem justified:

1. That in children the diagnosis frequently is not made until late: this has its influence on the type of operation to be selected.

2. That a preliminary thoracentesis should be done to ascertain the type of infection.

3. That in the streptococcus infections it is probable that aspiration or closed drainage through a tube would supply sufficient drainage and result in a lower mortality. These infections have a tendency to abort, recovery taking place without operation so that moderately good drainage will effect a cure where it would not in other types of cases.

4. That in cases of pneumococcus empyema, closed drainage is useful as a temporary measure in the case of the extremely sick patients but it is not to be recommended as the operation of choice. It does not supply adequate drainage and it does not allow the operator to free the lung, which can be done in a child through a small opening with one finger. This is a routine procedure with us and has a great deal to do with our scarcity of chronic cases. We would agree with Dowd that "if the lung expands at the time of operation, suction is not needed, and if it does not, suction will probably not make it."

5. That the causes of so-called collapsed lung is not the presence of atmospheric pressure through an open wound, but the result of inadequate operation and drainage. That in the few patients in whom it is unwise to free the lung at the first operation and in whom the lung remains bound down by adhesions, a decortication is required. This, in our experience, is always sufficient to obliterate the cavity and cause healing.

6. That operations causing collapse of the chest wall, as recommended by Estlander and Schede, are contraindicated because they cause distressing deformities and are unnecessary.

7. That there are certain definite indications for closed drainage and suction and this form of treatment should be limited to such cases only.

In conclusion, we would emphasize Hartwell's statement that, "no form of treatment for empyema which disregards the thorough drainage of the chest by a rib resection and the gradual re-expansion of the lung by respiratory efforts, meets the requirements."

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CHANGES IN THE FORM AND DIMENSIONS OF THE CHEST AT BIRTH AND IN THE NEONATAL PERIOD*

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In the fortnight following birth, the form and dimensions of the thorax undergo a series of peculiar changes which are dependent on a number of different factors. One of these changes, the increase in the horizontal circumference of the chest accompanying the first inspiration, is well known, but others, which are less marked and more variable, have apparently passed unnoticed. The following account of the changes in the form of the thorax of the new-born is based on measurements of the thoraces of late fetuses and full term still-born children, on observations of living infants on the first, third, fifth, seventh, tenth and twelfth days after birth, and on a number of observations on the horizontal chest circumference immediately before and after the first inspiration. The observations on the dimensions of the fetal thorax were made by Dr. L. A. Calkins as a part of an extensive study on the development of the proportions of the body in prenatal life, and our thanks are due him for permission to use these valuable data in the present investigations. The study on living infants was made at the Minneapolis General Hospital. In our study it proved impracticable to make thoracic measurements of the new-born until ten or fifteen minutes after birth and we have, therefore, made use of the excellent series of observations of Linzenmeier for data on the circumference of the chest immediately before and after the first inspiration.

Changes in the Horizontal Chest Circumference.—The horizontal circumference of the chest is usually determined at the level of the nipples, at the level of the lower end of the sternum, or at the tenth rib. Our study was based on measurements at the nipples and at the tenth rib. As the results of these measurements are not identical they will be considered separately.

The changes in the chest circumference at the level of the nipples are shown in Table 1 and in graphic form in Figures 1, 2 and 3. They are best presented by Figure 1, which is a semischematic curve show-

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1. Linzenmeier, G.: Der Verschluss des Ductus arteriosus Botalli nach der Geburt des Kindes, Ztschr. f. Geburtsh. u. Gynäk. **76**:242, 1914.

ing the entire series of modifications in this dimension from late fetal life until the close of the second week after birth. Four phases may be recognized in this curve. The first phase, which includes the latter part of fetal life, is characterized by a slow increase in chest circumference directly proportional to the growth of the body in length and without any marked changes in rate of growth. The second phase is inaugurated with the first inspiration. Within the first few minutes after birth, and often with the first breath, the chest cicumference increases about 2 cm., or approximately 8 per cent. This period of rapid increase is a transitory one for, as a rule, before the close of the first twelve hours a third phase, one of circumference decrease, begins and this continues for two or three days thereafter. The final phase, one of circumference recovery, sets in on the third or fourth day, and by the latter half of the second week the thorax has regained the circumference which obtains immediately after the first inspiration.

TABLE 1.—Changes in the Horizontal Circumference of the Chest (at the Level of the Nipples) at Birth and in the Neonatal Period

Age	Number of Cases	Average Circumference, Cm.	Average Circumference in Per Cent. of the Chest Circumference 15 Min. after Birth
Birth (before first inspiration)	40	29.3*	88.9
15 minutes after birth	36	31.2	100.0
12 hours after birth	36	30.7	92.8
3d day after birth	36	29.7	97.1
5th day after birth	34	30.2	97.7
7th day after birth	31	30.3	98.0
10th day after birth	19	30.5	98.8
12th day after birth	6	31.2	100.8

* Calculated from the data of Linzenmeier.

The modifications in the circumference of the chest at the level of the tenth rib are shown in Table 2 and in Figures 2 and 3. In later fetal life the circumference at this level, like that at the nipples, increases slowly and steadily at a rate directly proportional to the growth of the body in length. We have no direct data on the changes in the tenth rib circumference with the establishment of respiration, but judging from the changes in the diameters of the chest at this level, there is some increase in circumference although not as much as at the level of the nipples. The later postnatal changes in the tenth rib circumference are very similar to those in the nipple circumference. From the first to the fourth or fifth day, there is a period of circumference decrease. This is followed by a second period of slow increase, the initial circumference following the establishment of respiration being regained about the tenth or twelfth day (Fig. 2).

The postnatal decrease in chest circumference does not seem to have been observed previously. This is not surprising, for the loss is generally

small and would hardly be noticed unless systematic measurements were made day by day throughout the neonatal period on a considerable series of cases. The variability in the horizontal chest circumference at birth is considerable. Taylor [2] found that the standard deviation of the circumference at the nipples was 12.2 mm. for males and 17.7 mm. for females. With this degree of variation it is probable that the postnatal decrease would be entirely masked in any series of measurements taken at random at birth and in the neonatal period. While the reduction in chest circumference is often small, it seems to be fairly constant. Our measurements show some reduction at the level of the nipples in thirty-three of the thirty-six cases studied, and at the tenth rib in thirty-

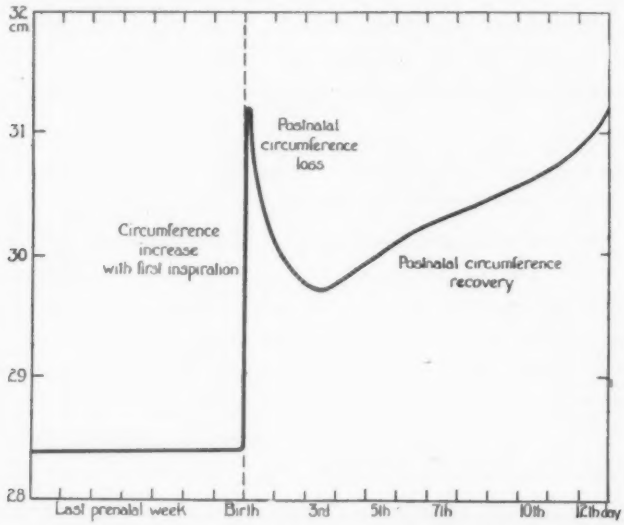

Fig. 1.—A semischematic curve illustrating the changes in the absolute circumference of the chest, at the level of the nipples, at birth, and in the first twelve days of postnatal life. Based on measurements of fetuses and still-born children and on observations on a series of living infants.

four cases. As is indicated in Tables 3 and 4, the variability in the amount of postnatal reduction is very great. The time of maximum reduction also differs considerably in various cases.

Since there is considerable individual variation in the circumference of the chest at birth, we have attempted to check the results of our absolute measurements by reducing these determinations to a percentage basis. This we have done by considering the circumference of the chest immediately after the establishment of respiration as 100 and calculating all later measurements as percentages of this initial one. This cal-

2. Taylor, R.: The Measurements of Two Hundred and Fifty Full-Term New-Born Infants, Am. J. Dis. Child. **27**:353 (April) 1919.

culation was first made for each case separately and the individual percentages for each period of observation were then averaged. This procedure eliminates much of the error which is introduced in the average absolute dimensions by initial differences in size of the individual cases. The results of these calculations are given in the fourth columns of Tables 1 and 2 and are illustrated by the curves in Figure 3. These figures on the *percentage* loss and gain of the chest circumference in the neonatal period differ slightly from those obtained from the averages of the absolute dimensions, but the general courses of the changes as determined by these two methods are in close agreement.

The causes of these changes in chest circumference seem to be diverse. The increase in the later part of fetal life reflects the general growth of the tissues and organs of the thorax and probably, in particular, the formation of new lung alveoli, which is very active in this period. The sudden increase at birth no doubt corresponds, in the main, to the initial enlargement of the thorax which takes place at the establishment of respiration and which is accompanied by the partial

TABLE 2.—CHANGES IN THE HORIZONTAL CHEST CIRCUMFERENCE (AT THE LEVEL OF THE TENTH RIB) IN THE NEONATAL PERIOD

Age	Numbef of Cases	Average Circumference, Cm.	Average Circumference in Per Cent. of the Chest Girth Immediately after Birth
15 minutes after birth.....	36	31.4	100.0
12 hours after birth.....	36	30.5	97.4
3d day after birth.....	36	29.5	96.6
5th day after birth.....	34	30.5	96.8
7th day after birth.....	31	30.8	97.8
10th day after birth.....	17	30.7	97.6
12th day after birth.....	11	31.0	100.9

expansion of the lung alveoli. It is possible that the dilation of the vascular bed of the lungs, following the close of the ductus arteriosus and the complete establishment of the pulmonary circulation, is also represented in this increase. The postnatal loss in circumference, on the other hand, seems to have nothing to do with the expansion of the lungs. In all probability, it is to be interpreted as another manifestation of the postnatal loss in mass of the infant, and, particularly, as an indication of the loss of fluid from the superficial tissues of the chest wall.

The changes following the phase of postnatal circumference decrease seem dependent on several factors. The enlargement of the thorax with the completion of the expansion of the lungs is probably the most important of these. The studies of a number of observers, and, particularly, the extensive observations of Gedgovd [3] have shown that all parts of the lungs are not fully expanded at the first inspiration or

3. Gedgovd, V. A.: Anatomical Peculiarities of the Respiratory Organs in Childhood. Diss. St. Petersburg, 1900.

even in the first few days, but that it is often a week or more before all of the alveoli are even partially dilated. And the investigations of Ridella,[4] as well as the older data of de la Croix [5] and Rossignol [6] indicate that this initial dilatation of the alveoli is not a complete one. These conclusions are supported by the careful micrometric studies on the lungs of fetal and newlborn animals by Addison and Howe.[7] It seems, then, that the volume of the lungs (and, therefore, the internal dimensions of the thorax) are normally increasing throughout the neonatal period, but that the effect of this increase on the external dimensions of the thorax is obscured in the first few days after birth by the more extensive decrease in the superficial soft parts of the chest wall which

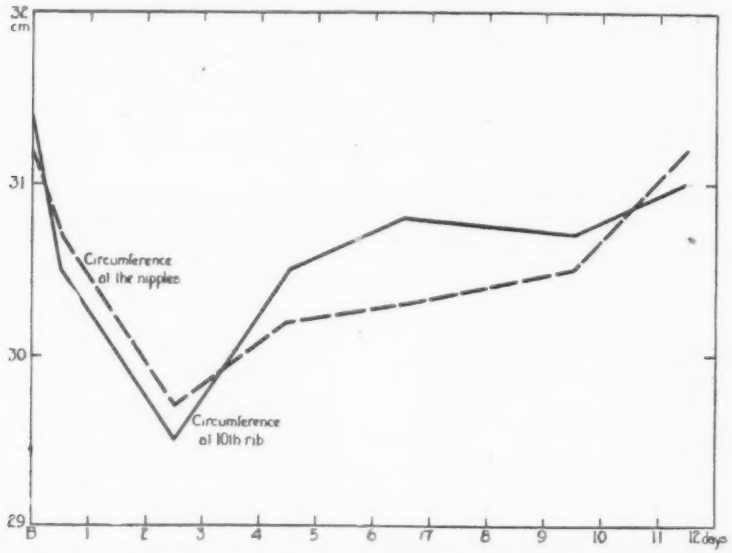

Fig. 2.—A graph illustrating the changes in absolute chest circumference, at the nipples and at the tenth rib, in the first twelve days of postnatal life. Based on measurements of a series of thirty-six children.

is associated with the postnatal weight loss. The effect of the continued increase in the volume of the lungs and thoracic cavity is pictured in the rise of the curve of the chest circumference which occurs in the latter part of the neonatal period after the major portion of the post-

4. Ridella, A.: Modificazioni che avvengo nel polmone prima o dopo la nascita in relazione colla funzione respiratoria, Folia Gynaecol. **7**:421, 1912.

5. de la Croix, N. J.: Die Entwicklung des Lungenepithels beim menschlichen Fötus und der Einfluss der Atmung auf Dasselbe, Arch. f. mikr. Anat. **22**:93, 1883.

6. Rossignol: Recherches sur la structure intime du pulmon de l'homme, Thèse, Bruxelles, 1846.

7. Addison, W. H. F., and Howe, H. W.: On the Prenatal and Neonatal Lung, Am. J. Anat. **15**:199, 1913.

natal weight decrease has taken place or when the recovery in body weight begins. A comparison of the changes in chest circumference with those in body weight in certain of our cases seems to support this view, for we find that the circumference loss and body weight loss are often synchronous while the recovery of the initial chest circumference often does not coincide with the recovery in body weight, but takes place a little sooner or even while the body weight is stationary. Other minor factors, such as the enlargement of the mammary glands associated with the formation of "witches' milk," may also have a slight effect on the circumference at the nipples.

TABLE 3.—Extent of Postnatal Loss in Chest Circumference at the Level of the Nipples. (Calculated in Per Cent. of the Chest Circumference 15 Minutes After Birth.)

	Number	Per Cent.
Cases showing no loss in circumference	3	9
Cases showing a loss of less than 5 per cent.	14	39
Cases showing a loss of 5 to 10 per cent.	14	39
Cases showing a loss of 10 to 15 per cent.	4	11
Cases showing a loss of 15 per cent. or more	1	2

The postnatal changes in the circumference at the tenth rib are probably due, in the main, to the same factors which influence the circumference at the nipples. The neonatal reduction at the tenth rib is a little greater and the recovery a little slower than at the nipples, presumably because the enlargement of the lower part of the chest in connection with the expansion of the lungs is less marked. Another factor which may play a part in the reduction of the tenth rib circumference is the postnatal decrease in the size of the liver which has been described by Miura.[8] This author finds a decline in the mass of the liver after birth which may be attributed to the decrease in blood content of the liver following the interruption of the flow of placental blood to the organ with the closure of the umbilical vein.

TABLE 4.—Extent of Postnatal Loss in Chest Circumference at the Level of the Tenth Rib. (Calculated in Per Cent. of the Chest Circumference 15 Minutes After Birth.)

	Number	Per Cent.
Cases showing no loss in circumference	2	6
Cases showing a loss of less than 5 per cent.	10	27
Cases showing a loss of 5 to 10 per cent.	20	56
Cases showing a loss of 10 to 15 per cent.	4	11

Changes in the Diameters of the Chest.—The anteroposterior and transverse diameters of the thorax were measured at the nipples and at

8. Miura, M.: Die Gewichtsabnahme verschiedener Organe vom Neugeborenen, Notizen meiner physiologischen und pathologischen Forschung, No. 15, 1912.

the tenth rib in our series of living children. The summarized results of these measurements are shown in Tables 5, 6, and 7 and are represented graphically by the curves in Figures 4 and 5.

The changes in the diameters of the chest at the level of the nipples and at the tenth rib are quite similar in the neonatal period. After breathing begins the anteroposterior and transverse diameters undergo a series of modifications in which three main phases may be recognized. Of these the first is a period of diameter decrease which begins after respiration is established and extends from twelve hours to three or four days thereafter. The second phase occupies the latter part of the first week and the beginning of the second. During this time the diameters

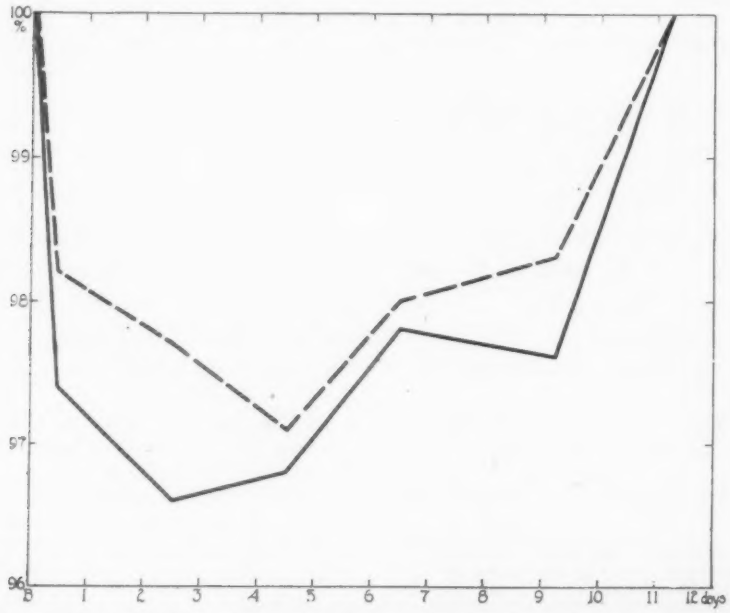

Fig. 3.—A graph illustrating the changes in the circumference of the chest at the level of the nipples and the level of the tenth rib. The circumferences for the first and later days are calculated in per cent. of the circumference of the chest ten to fifteen minutes after birth.

may decline slightly, remain stationary, or show minor fluctuations which are probably without significance. In the third period, which falls in the middle or latter part of the second week, the diameters again begin to increase and their initial dimensions (as observed immediately after the establishment of respiration) are generally regained between the tenth and fourteenth day. Thus the changes in the diameters of the thorax in the neonatal period show the same general characters of postnatal loss and recovery which were found in connection with the circumferences of the chest.

The postnatal reduction in the diameters of the chest is a fairly constant phenomenon. The anteroposterior diameter at the nipples showed a reduction in twenty-two of the twenty-three cases studied, and the transverse diameters in twenty cases. At the tenth rib the anteroposterior diameter was reduced in eighteen of twenty-three cases and the transverse diameter in twenty-two cases. The amount of reduction of the anteroposterior diameters is much greater both absolutely and relatively than the reduction of the transverse diameters. This is true of the diameters both at the level of the nipples and at the tenth rib. This difference, as well as the variation in reduction in individual cases, is shown in Tables 6 and 7. The causes of these changes will be considered in connection with the thoracic index.

Changes in the Thoracic Index.—The thoracic index is a value determined by dividing the anteroposterior diameter of the chest by the transverse diameter and multiplying the quotient by 100. The result

TABLE 5.—CHANGES IN THE DIAMETERS OF THE CHEST IN THE NEONATAL PERIOD

Age	Number of Cases	Average Diameters at the Nipples		Average Diameters at the Tenth Rib	
		Transverse	Anteroposterior	Transverse	Anteroposterior
15 minutes after birth	23	7.7	8.2	8.9	8.5
12 hours after birth	23	7.7	7.9	8.7	8.0
3d day after birth	23	7.5	7.7	8.8	7.9
5th day after birth	21	7.5	7.6	8.6	7.8
7th day after birth	18	7.5	7.7	8.8	8.0
10th to 12th days after birth	10	7.7	7.8	8.9	8.1

gives the anteroposterior diameter of the thorax in the form of a per cent. of the transverse diameter, and is an expression of the relative depth of the chest. Thanks to the studies of Hutchinson,[9] Mehnert,[10] Rodes,[11] Jackson [12] and others the general course of the changes in the thoracic index in the developmental period is well known. It is very high (nearly 200) in the young embryo because of the large size and great anteroposterior diameter of the heart and liver; but with the development of the thoracic skeleton it gradually falls to less than 90 in the fetus at term. In childhood the index continues to decline, usually falling to about 70 in maturity. The index is markedly affected at birth by a number of factors. The course of the modifications of the

9. Hutchinson, W.: Some Deformities of the Chest in the Light of Its Ancestry and Growth, J. A. M. A. **29**:512, 1897.

10. Mehnert, E.: Ueber topographische Altersveränderungen des Atmungsapparates, Jena, 1901.

11. Rodes, C. B.: The Thoracic Index in the Negro, Ztschr. f. Morphol. u. Anthropol. **9**:102, 1906.

12. Jackson, C. M.: Is Gravity the Factor Determining the Thoracic Index? Ztschr. f. Morphol. u. Anthropol. **10**:240, 1907.

thoracic index at the nipples in the neonatal period is shown in Table 8 and by Figure 6.

The changes in the index at the level of the nipples are shown in semischematic form in curve B of Figure 6. In the latter part of fetal life (ninth and tenth fetal months) the average index is about 86.[13]

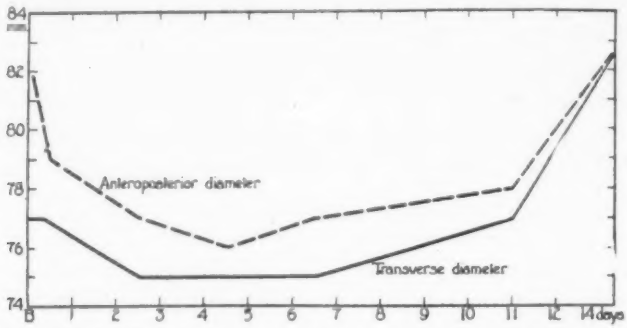

Fig. 4.—A graph illustrating the changes in the anteroposterior and transverse diameters of the chest, at the nipples, in the first twelve days after birth.

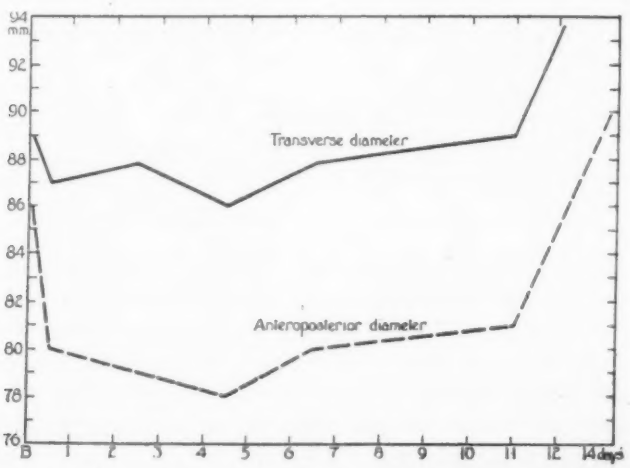

Fig. 5.—A graph illustrating the changes in the anteroposterior and transverse diameters of the chest at the tenth rib, in the first twelve days after birth.

At birth, with the establishment of respiration, it rises immediately to an average of 106. This initial rise is followed almost at once by a rapid decline, the index falling to an average of 102 within twelve hours after birth. During the remainder of the neonatal period, the measure drops

13. This determination was made from Dr. Calkins' material and is not corrected for the changes which occur in the chest on injecting the cadaver. Dr. Calkins' observations show that these changes tend to increase the anteroposterior diameter more than the transverse, and that the true thoracic index in later fetal life is a little lower than is here recorded.

slowly and irregularly, being about 100.5 in the middle of the second postnatal week. Six months or more pass before it has declined to the average which obtains just before birth.

The changes in the index at the level of the tenth rib are similar in character but are less pronounced than those at the level of the nipple. They are presented semidiagrammatically by curve A of Figure 6. In the latter part of fetal life and in children who have not breathed, the average index at the lower level is 91 or 92. With the establishment of respiration, it rises to an average of 95 and then declines again, in the course of the first twenty-four hours, to 92. By the middle of the second postnatal week it has dropped to 86.

TABLE 6.—Extent of Postnatal Loss in the Transverse and Antero-posterior Diameters of the Chest at the Level of the Nipples

	Antero-posterior Diameter	Trans-verse Diameter
Number of cases showing no reduction	1	3
Number of cases showing a reduction of less than 0.5 cm.	1	8
Number of cases showing a reduction of between 0.5 and 1.0 cm.	9	8
Number of cases showing a reduction of between 1.0 and 1.5 cm.	11	4
Number of cases showing a reduction of between 1.5 and 2.0 cm.	1	0

The rise in the thoracic index which occurs immediately after birth presumably takes place at the first breath when the chest wall is carried upward and forward as in the inspiratory movements of later life. The causes of the decline of the index which follows this initial rise are more obscure. We are inclined to attribute the change, in part at least, to the effect of gravity on the form of the chest. During prenatal life the fetus

TABLE 7.—Extent of Postnatal Loss in the Transverse and Antero-posterior Diameters of the Chest at the Level of the Tenth Rib

	Antero-posterior Diameter	Trans-verse Diameter
Number of cases showing no reduction	1	5
Number of cases showing a reduction of less than 0.5 cm.	2	8
Number of cases showing a reduction of between 0.5 and 1.0 cm.	8	5
Number of cases showing a reduction of between 1.0 and 1.5 cm.	9	4
Number of cases showing a reduction of between 1.5 and 2.0 cm.	3	1

is surrounded with fluid and consequently all external pressures are transmitted to its surfaces equally from all sides. Thus the tendency of all external pressures would be to mold the body into spherical form of which all diameters would be equal. But after birth, when the infant spends the greater part of the time prone on the back or abdomen, the force of gravity would be exerted on the anterior or posterior surfaces of the thorax, tending to decrease its anteroposterior diameter and to increase its transverse diameter. While it seems reasonable to regard

gravity as one of the important factors in the early reduction of the thoracic index, it is probable that the modifications in the forms of the chest which are due to this factor take place in a comparatively short time after birth. Jackson's experiments [12] indicate that gravity has little to do with the slow flattening of the chest which goes on in later infancy and childhood.

It is possible also that the dropping of the dome of the diaphragm after birth may have some effect on the thoracic index. That the

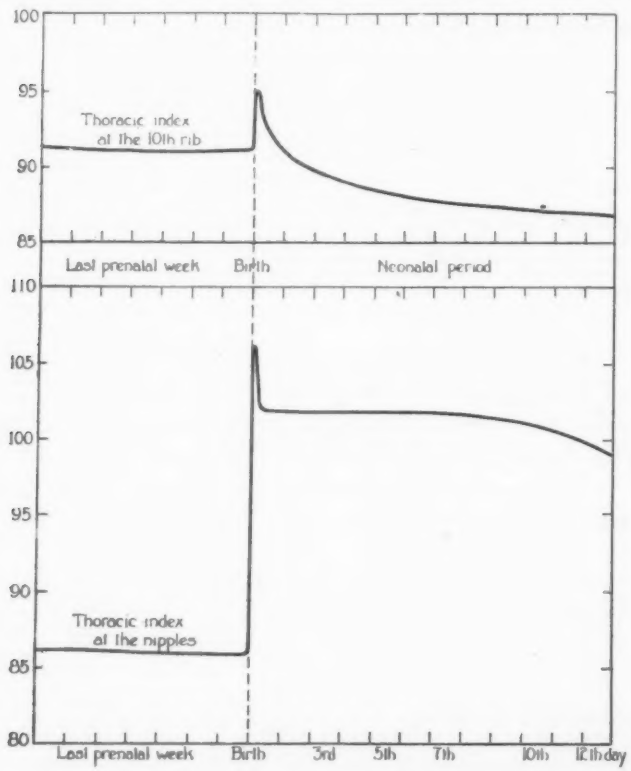

Fig. 6.—Two semischematic graphs illustrating the changes in the thoracic index at birth and in the neonatal period.

diaphragm does descend in the thorax shortly after birth seems well established, but so little is known either of the extent or the time of this change that it is hardly possible to correlate it with the modifications in chest form.

Whatever factors may be responsible for the modifications in the thoracic index in the neonatal period, these changes are correlated in an interesting manner with the internal changes of the lungs. It was

pointed out by Champneys [13] a number of years ago that the inflation of the lungs does not take place at the same rate in all parts of these organs. He found that the anterior surfaces and medial margins were expanded distinctly before the lateral portions and the inferior margins.[15] Thus the early expansion of the anterior surfaces and medial margins of the lungs reflects the early increase in the anteroposterior diameter of the thorax, particularly at the level of the nipples, while the subsequent expansion of the lateral portions corresponds to the later increase in the transverse diameters. And, similarly, the expansion of the lower portions and inferior margins is considerably delayed in correspondence with the slower and smaller changes in the circumference and thoracic index at the level of the tenth rib.

SUMMARY

At birth and in the neonatal period the thorax undergoes a series of modifications in dimensions and form which may be summarized as follows:

TABLE 8.—CHANGES IN THE THORACIC INDEX AT BIRTH AND IN THE NEONATAL PERIOD

Age	Number of Cases	Average of Indices	
		At the Level of the Nipples	At the Level of the Tenth Rib
9th fetal month	18	86.5	92
10th fetal month	13	86.0	92
Birth (full-term fetuses)	19	86.0	91
15 minutes after birth	23	106.0	95
1st day (12 hours) after birth	23	102.0	92
3d day after birth	23	102.0	89
5th day after birth	21	100.5	91
7th day after birth	18	102.0	91
10th and 12th days after birth	10	100.5	88

1. The horizontal chest circumference (at the nipples) is markedly increased at the first inspiration. In the first day, within twelve hours after birth, it enters a period of decrease which continues for two or three days and which is followed by a period of circumference increase. The initial circumference following the first inspiration is regained in the second week.

2. No data are available as to the changes in the circumference of the chest at the tenth rib with the establishment of respiration, although it is probable that they are somewhat similar to those at the nipples,

14. Champneys, F. H.: Second Communication on Artificial Respiration in Still-Born Children. The Expansibility of the Various Parts of the Lungs, Medico-Chir. Trans., Ser. 2 **42**:87, 1881.

15. These findings are substantially confirmed by G. J. Noback, working in this laboratory, who has investigated the condition of the thoracic viscera of the new-born both in the fresh condition and after intravascular fixation. His results will be published separately.

although less extensive. After respiration is established, the circumference at the tenth rib undergoes, first, a decrease, and then an increase, in a manner similar to that of the circumference at the level of the nipples.

3. The anteroposterior and the transverse diameters of the thorax, both at the level of the nipples and at the level of the tenth rib, show changes comparable with those of the circumferences at these levels. They decline in magnitude from the first to the fifth day after birth, and then enter a period of recovery, the initial dimensions (after respiration is established) being regained about the middle of the second postnatal week. The anteroposterior diameters undergo a much greater reduction than the transverse diameters.

4. The thoracic index at the nipples stands at about 86 before birth. With the establishment of respiration it rises to an average of 106 and then drops to about 102 in the first twenty-four hours. During the remainder of the neonatal period, it declines irregularly and slowly, being 100.5 in the middle of the second week. The changes in the thoracic index at the level of the tenth rib are of the same general character as those at the level of the nipples but are less pronounced.

5. The changes in the form and dimensions of the chest in the neonatal period (with the exception of the postnatal loss in circumference and diameter which seem to be associated with postnatal weight loss) are reflected in the order and degree of expansion of the different parts of the lungs.

THE ABSORPTION OF FLUID INJECTED INTO THE PERITONEAL CAVITY*

B. S. DENZER, M.D., AND A. F. ANDERSON, M.D.
NEW YORK

Intraperitoneal injection is one of the accepted methods of parenteral administration of fluid. In conditions such as diarrhea, in which dehydration is extreme, repeated administration of fluid is often necessary—under the skin, into the veins and into the peritoneal cavity. The question of the absorption of fluid by the peritoneum is, therefore, a matter of very practical import.

Postmortem examination of infants that received fluid intraperitoneally a short time before death, has already thrown some light on the problem of absorption. Blackfan and Maxcy [1] conclude that absorption is rapid. They record the recovery of 20 c.c. of fluid in an infant weighing 20 pounds, that received 250 c.c. eighteen hours, and 200 c.c. six hours before death. The fluid was clear yellow; the peritoneum was glistening and no mention was made of deposition of fibrin, cellular exudate or other evidence of inflammatory reaction.

Our own necropsy findings indicate varying rates of absorption. (Table 1). In several cases, large amounts of fluid injected within thirty hours before death were completely absorbed; occasionally fluid was present at necropsy several days after injection, and in two cases fibrin was found in the iliac fossae. As a rule, all but a comparatively small amount of the injected fluid was absorbed at the end of twenty-four hours.

The use of capillary needles for abdominal puncture described recently [2] provided a method of tracing the reaction to and the absorption of injected fluids. During the course of the work the technic and instruments were modified.

The original method consisted in the use of a metal needle to puncture the skin, and inserting through the opening thus made a glass capillary needle. To obviate the disadvantage of using glass needles, metal cannulas and glass capillary tubes were devised. Only the latest and thus far the most satisfactory type will be described.

The instrument consists of a trocar-cannula [3] and glass capillary tubing and is shown in Figure 1. The cannula is conveniently held

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* From the Children's Service of the New York Nursery and Child's Hospital (Dr. Oscar M. Schloss, Director), and the Department of Pediatrics, Cornell University Medical College.

1. Blackfan and Maxcy: Am. J. Dis. Child. **15**:19 (Jan.) 1918.
2. Denzer: Am. J. Dis. Child. **20**:113 (Aug.) 1920.
3. The instruments were made for us by the Randall Faichney Co. of Boston.

by handles on either side. The shoulder is as short as it can be made, and is so constructed that on removal of the trocar it will take a Luer syringe or a Luer adapter. The object of this is to permit the injection of saline solution following the test puncture for the presence of fluid. The shaft of the cannula is 17 gage and one half inch in length. The point of the cannula is made in two forms (Fig. 1 A). One is blunt; therefore the trocar is sharp and bevelled in the form of a spear

TABLE 1.—POSTMORTEM FINDINGS IN TEN CASES IN WHICH FLUID WAS INJECTED INTO THE PERITONEAL CAVITY

Name	Age	Weight Lbs. Oz.	Diagnosis	Amount Injected, C.c.	Interval Between Injection and Death	Amount of Peritoneal Fluid Found Postmortem, C.c.	Remarks
Stanley G. ...	5 mos.	13 ..	Acute intestinal intoxication	150	2 hrs.	60	Turbid fluid; plaques of epithelial cells; many degenerated
L. S.	5 mos.	11 ..	Acute intestinal intoxication	250	1½ hrs.	150	
John T.	7 mos.	10 ..	Acute intestinal intoxication	150	20 hrs.	½	16,000 cells per c.mm.; lymphocytes predominant; many degenerated
Walter F. ...	13 mos.	18 ..	Pneumonia	200	16 hrs.	18	Yellow turbid fluid
Jane C.	8 mos.	17 5	Acute intestinal intoxication	275 250	30 hrs. 16 hrs.	none	
Julia M.	5 wks.	4 8	Pneumonia	125	40 hrs.	none	
Dalton D. ...	3 mos.	6 ..	Acute intestinal intoxication	100	28 hrs.	none	
Wm. W.	10 wks.	7 4	Acute intestinal intoxication, bronchopneumonia	130	4 days	4	
Henry B.	3 mos.	6 ..	Acute intestinal intoxication	100 125	53 hrs. 34 hrs.	15	Intestinal coils edematous, covered with sticky mucoid material; 2 pieces yellowish fibrin 2.5 cm. long in iliac fossa
Catherine S.	2 mos.	6 ..	Acute intestinal intoxication, bronchopneumonia	175	69 hrs.	4	Few flakes of fibrin in iliac fossa

head. This type of trocar-cannula requires more pressure to insert it through the abdominal wall, but on removal of the trocar, it has the advantage—possibly only theoretical—of presenting a flat surface to the intestinal coils. The second form of cannula has a sharp bevelled tip (Fig. 1 B), and the point of the trocar is made to correspond. The advantage of this form of cannula is its easy insertion through the abdominal wall; and an objection—possibly only theoretical—is that it presents a sharp point to the intestinal coils.

Most of the capillary tubes were prepared by us from tubing about 4 mm. in diameter. This tubing was drawn out in the Bunsen flame and, naturally, varied in thickness. Only those parts were used that accurately fitted the shoulder or rather the proximal part of the shaft, and the tubing was cut off so as to protrude 1 or 2 mm. from the tip of the cannula. The purpose of this technic is to prevent the breaking of the capillary tube within the peritoneal cavity. The cannula with the capillary tube prepared in this way is illustrated in Figure 1 C. The capillary tube impinges on the wall of the cannula at (*a*). After the tubing is safely inserted into the cannula, a break, if it occurs at all, will take place above the point—(*a*) and the lower fragment, firmly fixed in the shaft, can be withdrawn with the cannula. During the latter part of this study, capillary tubing was prepared by glass blowers (Fig. 1 D).

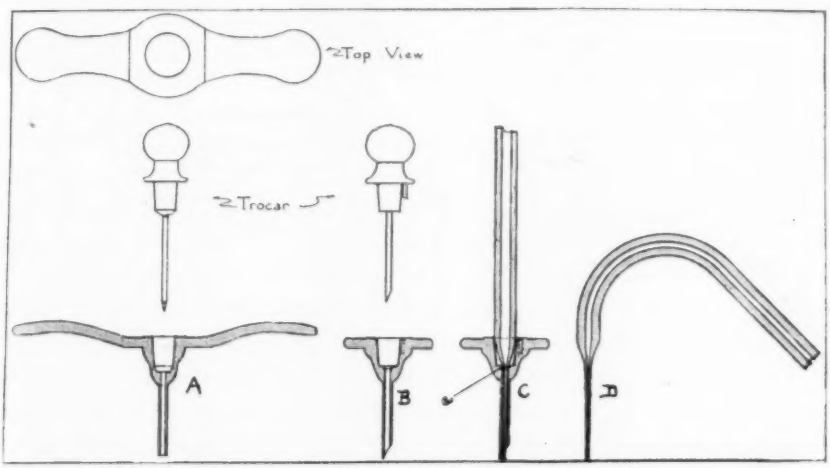

Fig. 1 (Drawn to scale and double the actual size).—A. Cannula with blunt tip and trocar with spear head point. B. Cannula and trocar with bevelled point. C. Cross section of cannula with tubing in place. Tubing is firmly fixed in the cannula at *a*. D. Tubing drawn out by glass blowers.

When glass tubing was used repeatedly, the utmost precautions were necessary to obtain clean surfaces. As capillarity depends on the capacity of the liquid to moisten the surface of the tube, a minute film of fatty material might check the rising column of fluid. Cleaning fluid, hot water, alcohol and ether were used in obtaining clean glass ware.

The technic with the trocar-cannula and capillary tube is as follows: After sterilization of the skin and the usual precautions to determine bladder distention, the trocar-cannula is inserted; the sudden release of pressure indicates that the peritoneal cavity has been entered. The

trocar is then removed, and the capillary tube is inserted as far as it will go. It is advisable to wait a few moments and to turn the needle in various directions before concluding that there is no fluid. Possibly, a bit of omentum or the wall of the intestine may temporarily block the capillary tube; or, when there is only a small amount of fluid, this momentary wait and the straining and crying of the infant

TABLE 2.—RESULTS OF ABDOMINAL TAP IN CASES NOT RECEIVING INTRAPERITONEAL INJECTIONS OF FLUID

Name	Age	Weight, Lbs.	Weight, Oz.	Diagnosis	Result of Tap*
Dalton D.	10 weeks	6	11	Intoxication	0
Wm. N.	4 months	9	6	Intoxication	0
Thos. B.	3 months	6	..	Intoxication	0
Edward D.	10 weeks	6	10	Intoxication	0
Veronica H.	10 months	13	8	Intoxication	0
Margaret C.	8 months	15	6	Intoxication	0
Wm. J.	8 months	12	2	Tuberculosis	0
Baby Gon.	3 weeks	6	6	Septicemia	0
Gordon G.	10 months	12	..	Malnutrition (distention)	0
Daniel	4 weeks	6	..	Malnutrition	0
Wm. W.	3 weeks	6	..	Malnutrition	0
James B.	3 months	6	2	Marasmus	0
Mary S.	3 months	8	..	Intoxication	0
Raymond A.	8 months	10	14	Marasmus	0
John G.	6 months	13	..	Intestinal indigestion	0
Drouet	4 months	..	..	Marasmus	0
Marie C.	4 weeks	6	8	Intestinal indigestion	0
Paulo R.	2 months	8	12	Intoxication, marasmus	0
Frank S.	11 weeks	..	..	Marasmus	0
LeRoy C.	4 weeks	8	1	Intestinal indigestion, syphilis	0
Mary C.	5 months	7	2	Intestinal indigestion	0
Madeleine E.	4 months	7	4	Intestinal indigestion	0
John M.	10 weeks	5	6	Marasmus	0
Hannah R.	10 weeks	8	4	Malnutrition	0
Stewart S.	5 months	11	8	Rickets	0
William W.	5 months	12	6	Rickets	0
Susan H.	4 months	8	2	Tuberculosis	0
Lucinda P.	3 months	5	8	Marasmus	0
Dominick D.	5 months	9	8	Pneumonia	0
Martha L.	4 months	9	..	Malnutrition	0
Richard P.	10 weeks	5	12	Malnutrition	0
John S.	5 months	8	13	Intoxication	0
Virginia K.	4 months	4	..	Marasmus	0
John R.	4 months	11	8	Intoxication	0
Agnes H.	3 months	6	..	Marasmus	+
Joseph W.	18 months	11	2	Marasmus, tuberculosis	+
James P.	3 months	5	8	Marasmus	+
Joseph B.	7 months	5	14	Marasmus	+
Marie O'D.	6 months	8	8	Rickets	+
Isaac S.	11 months	14	14	Rickets	+
Lloyd L.	8 months	9	13	Rickets	+
Leon T.	4 months	9	12	Rickets	+
Herman B.	6 months	16	..	Rickets, tuberculosis	+
Edward T.	3 years	..	..	Peritonitis, tuberculosis	+
Baby L.	2 months	6	6	Peritonitis	+
Pietro V.	2 months	10	..	Empyema, peritonitis	+
Beatrice J.	.....	..	..	Tuberculosis	+
May S.	.....	..	..	Tuberculosis	+
Hugh R.	14 months	14	12	Tuberculosis	+
Baby H.	.....	..	..	Cirrhosis luetic	+

* 0 = dry tap, + = fluid obtained.

may aid in distributing it over the entire peritoneal cavity. At any rate, the sight of the gradually rising column of fluid has frequently rewarded this precaution. If the tap is dry and an injection of saline solution is indicated, it may be given through the cannula. Any place on the abdomen, except those parts covering a solid viscus or one

fixed by a short mesentery, may be used. As a rule, punctures were made a short distance below the umbilicus in the midline.

Abdominal taps have been made in fifty cases (Table 2). In thirty-four cases no fluid was obtained. The sixteen cases which showed fluid comprised three cases of generalized tuberculosis, one of acites with congenital syphilis, three of peritonitis, four of marasmus and five of rickets with pot belly.[4] We may conclude, then, that in the normal peritoneal cavity no free fluid can be demonstrated by the method employed by us.

It was important to determine whether fluid can be obtained with the capillary tube only when there is a large ascites or whether this method can demonstrate very minute quantities. The effort was, therefore, made to reproduce a very small peritoneal effusion and then to recover some of the fluid with the capillary tube.

A puncture in the midline of the abdomen was done on Baby M., a marantic infant, 10 weeks of age. As no fluid was obtained, 15 c.c. of Ringer's solution was injected through the needle which was left in situ. The child was turned in various positions to produce an even distribution of fluid, and after an interval of five minutes, the capillary tube was inserted and fluid obtained. The baby was again turned in various positions, a second interval of five minutes elapsed and fluid was again obtained. The child was then given an intraperitoneal injection of Ringer's solution.

A similar procedure was adopted in the case of Baby P., an ill nourished infant, 3 months of age. However, this child received only 5 c.c. of Ringer's solution. Nevertheless, five minutes after the injection, fluid was obtained with the capillary tube.

In these two cases fluid was withdrawn at the same site at which it was injected. It is conceivable that the omentum or loops of intestine might temporarily have walled off the injected fluid; thus the small effusion characterized by the even distribution of the fluid over the entire peritoneal cavity, was not faithfully reduplicated. The procedure was therefore modified. In the next case, Baby H. R., an ill-nourished infant, 10 weeks old, an abdominal puncture was done in the left lower quadrant, 4½ cm. from the midline. No fluid was obtained, and 5 c.c. of Ringer's solution was injected through the needle which was then withdrawn. Seven minutes later, the abdomen was tapped just below the umbilicus and fluid was obtained. The intraperitoneal injection of Ringer's solution was then completed. This indicates that there is a thorough distribution of small quantities of fluid throughout the

4. The cases of peritonitis will be the subject of a later communication and the incidence of free fluid in marasmus is under investigation.

peritoneal cavity, and that extremely small amounts may be demonstrated by the capillary tube.

The rate of absorption of fluid injected into the peritoneal cavity was studied in two series of cases. In the first series, single taps were done a number of hours after one or a series of intraperitoneal injections. Seven cases showed a dry tap from twelve to twenty-four hours after injection. In one case fluid was present twenty hours, and in two cases, twenty-four hours after injection (Table 3).

The second series comprised ten cases. After a preliminary dry tap, fluid was injected and abdominal punctures were performed at stated intervals thereafter. Absorption was variable—from twelve to forty-eight hours. Solutions of different composition were tried. In the first six cases, physiologic sodium chlorid solution or Ringer's solution was used. As injections into the serous cavities of animals

TABLE 3.—RESULTS OF SINGLE ABDOMINAL TAPS FOLLOWING INTRAPERITONEAL INJECTION OF FLUID

Name	Age	Weight Lbs.	Weight Oz.	Diagnosis	Hours After Intraperitoneal Injection	Result of Tap	Remarks
D. D.	10 wks.	6	11	Intoxication	12	0	
Wm. N.	4 mos.	9	6	Intoxication	24	0	
Thos. B.	3 mos.	6	..	Intoxication	18	0	
Ed. D.	10 wks.	6	10	Intoxication	24	0	
V. H.	10 mos.	13	8	Intoxication	12	0	
M. C.	8 mos.	15	6	Intoxication	20	0	
M. S.	3 mos.	8	..	Intoxication	24	0	
J. H.	3 mos.	6	6	Intoxication	24	+	16,000 cells per c.mm., culture negative
J. S.	6 mos.	12	4	Intoxication	20	+	15,000 cells per c.mm., culture negative
J. C.	8 mos.	19	2	Intoxication	24	+	12,600 cells per c.mm., culture negative

indicates a more rapid absorption of hypotonic solutions than of normal or hypertonic solutions,[5] a 0.3 per cent. sodium chlorid solution was used in four cases. In this small series, the rate of absorption of the hypotonic solution was not materially more rapid than that of other solutions. Clinical signs of a reaction, such as distention and slight rise in temperature, were identical with those following the injection of physiologic sodium chlorid solution. Although evidence of the distinct superiority of hypotonic solutions is lacking, in our brief experience there is no indication that such solutions are less safe than physiologic sodium chlorid solution (Table 4).

The reaction of the peritoneum was studied by tracing the total and differential cell count (Table 5; Fig. 2). The number of cells

5. Leathes, J. B., and Starling, E. H.: J. Physiol. **13**:106, 1895. Starling, E. H., and Tubby, A. H.: J. Physiol. **16**:140, 1894. LePlay, A., and May, E. S.: Compt. rend. Soc. de biol., Paris, T. 11, 221, 344. Adler, I., and Meltzer, S. J.: J. Exper. Med. **1**:482, 1896.

TABLE 4.—Results of Abdominal Taps at Intervals After Intraperitoneal Injection of Fluid

Name	Age	Weight Lbs.	Weight Oz.	Diagnosis	Preliminary Tap	Amount Injected, C.c.	Postinjection Taps Hours After Injection 3	6	12	24	36	48	Composition of Fluid Injected
L. C.	4 mos.	8	1	Syphilis, acute intestinal intoxication	0	150	+	+	+	+	0	..	Commercial tablets, Ringer's Sodium chlorid.....0.7 Potassium chlorid.....0.026 Calcium chlorid.....0.03
Wm. W.	3 wks.	6	9	Malnutrition	0	150	+	+	+	0	..	..	Commercial tablets, Ringer's Sodium chlorid.....0.7 Potassium chlorid.....0.026 Calcium chlorid.....0.03
M. C.	5 mos.	7	12	Acute intestinal intoxication	0	150	+	+	+	0	..	..	Prepared from chemically pure materials* Sodium chlorid, 0.85 per cent.
M. E.	4 mos.	7	4	Acute intestinal intoxication	0	150	+	+	0	..	..	..	Prepared from chemically pure materials* Sodium chlorid, 0.85 per cent.
J. M.	2½ mos.	5	6	Marasmus	0	65	..	+	+	+	+	0	Prepared from chemically pure materials* Sodium chlorid.....0.9 Calcium chlorid.....0.026 Potassium chlorid.....0.01
H. R.	2½ mos.	8	4	Malnutrition	0	150	..	+	+	+	+	0	Sodium chlorid.....0.7 Calcium chlorid.....0.026 Potassium chlorid.....0.01
M. L.	4 mos.	9	..	Malnutrition	0	100	..	..	+	0	..	..	Sodium chlorid, 0.3 per cent.
V. K.	4 mos.	4	..	Marasmus	0	100	..	..	+	+	0[1]	..	Sodium chlorid, 0.3 per cent.
J. T.	7 mos.	10	..	Acute intestinal intoxication	0	150	..	..	+	+[2]	..	..	Sodium chlorid, 0.3 per cent.
J. R.	4 mos.	11	8	Acute intestinal intoxication	0	100	..	..	+	0[3]	..	..	Sodium chlorid, 0.3 per cent.

* Merck's reagent chemicals.

1. Done 30 hours after injection.
2. Done at necropsy; ½ c.c. fluid obtained
3. Done 20 hours after injection

rises rapidly from the third and sixth hours to the thirty-sixth hour. At the peak of the reaction the count rose in most cases to 12,000 and in one case to 34,000. Not all fluids could be studied in this way. Some were too thick and mucoid, others clotted too quickly to permit dilution and the use of the counting chamber, and occasionally only a drop or two of fluid was obtained. Cultures done on twelve fluids were negative.

The differential smears were particularly interesting (Table 5). In one case, in the fresh preparation, large cells were seen putting out pseudopodia—true ameboid motion. The stained preparations of a number of the cases showed large vacuolated mononuclear cells con-

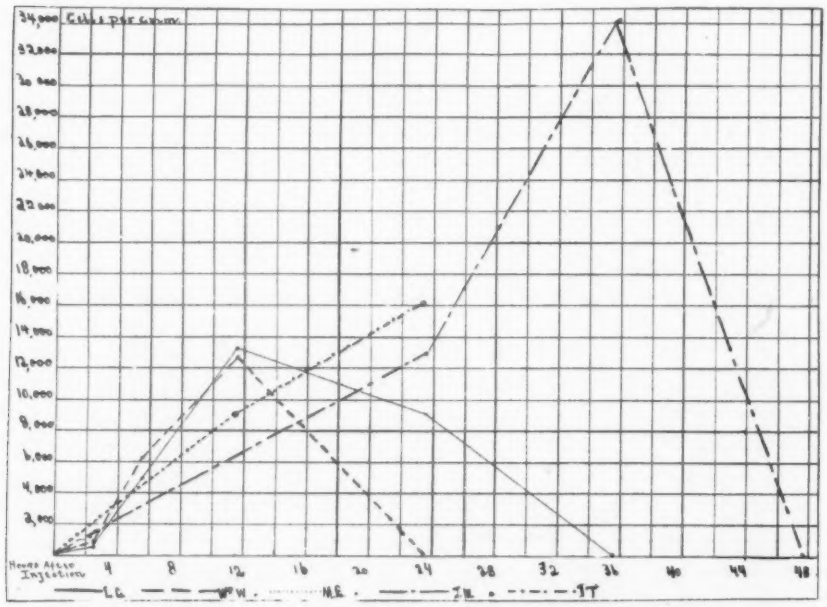

Fig. 2.—Curves showing reaction of peritoneum to injections as determined by leukocyte count.

taining leukocytes and free chromatin bodies—macrophages or endothelial cells of Metchnikoff. These cells, according to Adami, are characteristic of the early inflammatory reaction of the peritoneum.[6] The differential counts do not presume to represent the exact percentages of the different cells. The material as mentioned above, was thick and mucoid and did not lend itself to even distribution on a slide; also, many of the cells were in various stages of degeneration and others were distorted by the albuminous matrix and fibrinous strands. The essential fact is that the peritoneal fluid shows all the character-

6. Adami, J. G.: Inflammation, New York, Macmillan & Co., 1907, p. 69.

TABLE 5.—Reaction of Peritoneum as Traced by Leukocyte Count

Name	Hours After Injection										
	3		6		12		24		36		48
	Total Count	Differential	Total Count	Differential	Total Count	Differential	Total Count	Differential	Total Count	Differential	
L. C.	590	.....	4,800	.....	13,200	.....	9,000	.....	.....	0	
M. C.	720	.....	5,400	.....	.....	Not done	.....	0			
M. E.	700	.....	2,800	.....	.....	0					
Wm. W.	600	Poly. 87 Lym. 13	6,300	Poly. 91½ Lym. 8½	12,800	.....	.....	0			
J. M.	.....	.....	.....	Poly. 91 Lym. 7 Trans. 2	.....	Poly. 84 Lym. 8 Trans. 8	12,900	Poly. 59 Lym. 24 Endo. 13 Trans. 4	34,400	Poly. 68 Lym. 30 Endo. 1 Trans. 1	0
H. R.	.....	.....	.....	Fluid obtained unsatisfactory for total or differential count	.....	.....	.....	Poly. 67 Lym. 29 Endo. ½ Trans. 3½	.....	Poly. 60 Lym. 35 Endo. 3 Trans. 2	0
V. K.	.....	.....	.....	.....	5,000	Poly. 79 Lym. 20 Trans. 1	.....	Poly. 71 Lym. 24 Endo. 1	.....	0	
J. T.	.....	.....	.....	.....	9,000	Cells degenerated; unsatisfactory	16,000*	Postmortem unsatisfactory			
M. L.	.....	.....	.....	.....	.....	Poly. 93 Lym. 7	.....	0 †			
J. R.	.....	.....	.....	.....	Quantity insufficient	Poly. 79 Lym. 18 Endo. 1 Trans. 1	.....	0 †			

Poly. = polynuclear; Endo. = endothelial macrophages; Lym. = lymphocytes; Trans. = transitionals.
* Postmortem. † Done 20 hours after injection.

istics of an inflammatory reaction—mucoid, sticky, fibrinous fluid, containing endothelial cells and leukocytes—a true chemical peritonitis.[7]

The usual criterion by which one judges whether an intraperitoneal injection may be repeated, is the disappearance of the distention which usually follows injection. We have not kept notes as to the exact hour of the disappearance of this sign. However, in no case did distention persist for as long a period as fluid was demonstrable. The occurrence of a temporary inflammatory reaction and the persistence of a small residue of the previously injected fluid should not contraindicate the repetition of an injection.

SUMMARY

1. Absorption of fluid injected into the peritoneal cavity occurs in from twelve to forty-eight hours.

2. Intraperitoneal injection of saline solutions causes a temporary reaction—a sterile inflammation.

3. Abdominal puncture with a capillary tube provides a safe method for the diagnosis of very small quantities of peritoneal fluid and of following the course of an inflammatory reaction in the peritoneal cavity.

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7. In the early part of the work, this albuminous fluid rendered preparations stained by the usual methods very unsatisfactory. We wish to thank Dr. B. S. Kline for suggesting a method that produces excellent pictures. The technic is: Fixation in cold 10 per cent. liquor formaldehyd for ten minutes; washing in tap water and staining with aqueous or saturated alcoholic solution of methylene blue.

THE PHYSICAL DEVELOPMENT OF TUBERCULOUS CHILDREN *

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Deficiency in body weight is generally recognized as one of the important early symptoms of tuberculosis. In adults this is frequently synchronous with the appearance of focal symptoms. In childhood, focal symptoms play so unimportant a rôle in the early differential diagnosis of tuberculosis, and are so lacking in pathognomonic features, that it seems necessary to determine whether deficiency in weight can be considered of value.

Two groups of children form the basis of this study: the first group, those of nontuberculous parentage, who were themselves either without any symptoms suggestive of tuberculosis, or who had symptoms and were negative to tuberculin tests. This constituted the control group (614 children). This series was observed in the pediatric department of the West Side Jewish Aid Dispensary. The second group, comprising 508 patients, was observed in the course of a number of years at the West Side Jewish Aid Branch of the Municipal Tuberculosis Sanitarium. These children came under observation as the children of tuberculous parentage, as children suspected of tuberculosis because of poor physical condition, or referred to the tuberculosis department from the pediatric dispensary after a positive diagnosis had been made.

No attempt was made to differentiate between the various types and stages of tuberculous infection. Personal observation leads me to believe that tuberculosis in early childhood remains an acute disease over an undetermined number of years, and that there is no method which enables one to determine the activity or quiescence of the infection. The diagnosis of the tuberculosis was based on physical findings and the von Pirquet test. The physical findings were very largely those of bronchial and hilus gland involvement. This group of children, under observation for several years, was studied to see if a conclusion could be drawn as to the effect of a recognized tuberculous infection on the height and weight.

The majority of cases were tested with both bovine and human tuberculin. No case was recorded as positive unless it gave the characteristic reaction to tuberculin. This point is mentioned because it is

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*Read before the joint meeting of the Chicago Medical Society and Chicago Pediatric Society, Nov. 17, 1920.

my opinion that physical findings are not sufficient grounds for the diagnosis of tuberculosis in the absence of the positive tuberculin reaction (except when overwhelming infections with the tubercle bacillus are present, or the acute exanthemata, especially measles and scarlatina. It is also likely to be negative in pertussis and pneumonia). The basic clinical work of von Pirquet, the observations of Franz Hamburger, and the combined clinical and pathological study made by Gohn, prove conclusively that the infection may be demonstrated postmortem in cases in which the cutaneous test was positive during life.

In order to establish a foundation of normal height and weight of children in this district, a control group was used. The comparison of this group with the ideal weights given by the Children's Bureau of the Department of Labor, shows clearly the necessity for comparing special groups of children with like groups from the same social status, living in a similar environment. Only by such comparison is it possible to minimize errors in deduction.

I will first consider the statistics (Tables 1 to 6) which have to do with the initial height and weight of the boys and girls in the three groups, namely, Children's Bureau, Control and Tuberculous. From these tables, charts were plotted indicating graphically the measurements at half year periods, from 1 to 14 years, inclusive.

The tuberculous and control curves do not rise evenly like those of the Children's Bureau. The reason for this is that there was not the same number of observations in each age period, and the total number in each series was relatively small (tuberculous 508, control 614) in comparison with the thousands of children observed by the Children's Bureau.

It also appears more than a coincidence that the depressions and elevations of the tuberculous and control curves should so closely correspond as they do. The explanation seems to be that the children of these series came from a small district in which hygienic, dietetic, economic, and social conditions were decidedly adverse, so that while there is a well marked difference in their heights and weights, nevertheless their measurements rose and fell in rather parallel fashion.

The features that have been especially noted are: (1) The number of points at which the control and tuberculous lines have risen above the Bureau line, indicating the number of age groups that have been normal in the available statistics; (2) the number of individuals represented by these normal age groups, and (3) the percentage of the series that came up to the Bureau standard.

These three observations are represented in a column, indicative of the percentage of normality of the control and tuberculous groups in comparison with the Children's Bureau standard, represented as 100 per cent.

TABLE 1.—HEIGHT AND WEIGHT OF TUBERCULOUS BOYS (256)

Age. Years	Number of	Initial Measurements		Period of Observation in	Average Gain in This Period		Average Gain per Year While Observed	
		Height, Inches	Weight, Pounds		Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	1	....	20.2	35.0	....	15.8	....	5.4
1½	2	....	22.4	25.0	....	9.5	....	4.5
2	6	33.1	24.6	30 8	7.5	10.2	2.3	3.9
2½	6	35.7	28.7	27.6	7.4	9.9	3.2	4.3
3	4	37.0	31.6	50.0	10.2	20.4	2.4	4.8
3½	1	....	34.7	51.0	....	16.5	....	3.8
4	12	38.2	34.5	29.6	7.0	10.3	3.0	4.1
4½	2	40.3	31.5	14.0	1.7	3 0	1.4	2.5
5	15	41.0	38.4	35.3	6.8	14.6	2.2	4.9
5½	7	43.0	39.1	36.0	4.8	9.8	1.6	3.2
6	11	44.8	41.6	35.1	5.6	14.0	1.9	4.7
6½	2	....	43.3	51.5	....	21.2	....	4 9
7	30	46.1	45.9	24.3	5.5	16.5	1.9	5.8
7½	8	45.9	47.9	25.7	4 3	12.8	2.0	5.9
8	29	48.1	52.9	31.7	3.5	19.2	1.3	7.2
8½	2	45.0	50.7	15.0	....	24.5	....	19.6
9	24	49.2	57.2	24.5	4 6	13 0	2.2	6.3
9½	3	41.6	56.6	21.3	10.1	12 5	5.6	7.0
10	28	52.2	58.1	28.0	4.6	20.9	1.9	8.9
10½	1	....	67.5	29.0	....	23.5	....	9.7
11	12	51.9	61.1	17.9	4.3	12.2	2.8	8.1
11½	..	....	....	....	....	....	....	....
12	17	54.0	68.7	17.0	4.2	20.5	2.9	14.4
12½	7	53.8	73.3	14.0	6.3	12.3	5.3	10.5
13	13	55.1	75.6	19.7	4.1	14.2	2.4	8.6
13½	3	58.1	92.5	25.0	8.0	27.9	3.8	13.3
14	10	56.7	82.2	22.5	5.2	14.3	2.7	7.6

TABLE 2.—HEIGHT AND WEIGHT OF TUBERCULOUS GIRLS (252)

Age. Years	Number of	Initial Measurements		Period of Observation in	Average Gain in This Period		Average Gain per Year While Observed	
		Height, Inches	Weight, Pounds		Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	2	....	22.9	33.0	1.0	12.3	0.3	4.4
1½	2	....	20.5	16.0	....	5.0	....	3.7
2	6	32.1	24 4	23.0	3.8	8.1	1.9	4.2
2½	5	35.8	30.6	49.5	10.7	23.9	2.6	5.7
3	9	34.6	29.3	19.4	3.9	6.1	2.4	3.7
3½	1	38.0	32.5	23.0	15.2	6.7	7.9	3.4
4	11	39.5	32.8	33.0	5.7	15.0	2.0	5.4
4½	1	37.0	29.0	29.0	....	9.2	....	3.8
5	13	40.9	36.3	25.9	5.5	11.7	2.5	5.4
5½	2	41.0	35.9	21.0	2.3	8.2	1.3	4.6
6	19	42.9	42.0	27.4	3.8	10.1	1.6	4.4
6½	3	43.7	39.1	44.6	6.9	18.4	1.8	4.9
7	16	44.2	42.9	32.1	6.3	14.7	2.3	5.4
7½	1	....	46.5	69.0	....	51.2	....	8.9
8	19	47.4	49.7	34.9	5.9	19.6	2.0	6.7
8½	8	48.1	48.2	26.7	4.4	20.2	1.9	9.0
9	21	49.0	53.0	21.0	4.9	12.3	2.7	7.0
9½	3	49.8	56.1	27.0	10.2	15.4	4.5	6.8
10	26	50.1	58.2	26.6	5.0	20.3	2.2	9.1
10½	2	53.8	67.7	14.0	3.7	15.0	3.1	12.8
11	41	52.7	62.0	25.7	3.2	15.1	1.4	7.0
11½	..	....	....	....	....	....	....	....
12	14	57.5	68.7	28.0	1.6	20.7	0.6	8.8
12½	3	53.8	72.1	20.5	5.0	35.9	1.9	20.1
13	17	58.0	82.6	18.8	9.8	22.7	6.2	14.4
13½	2	61.7	77.0	18.0	1.5	12.2	1.0	8.1
14	5	52.6	83.1	9.0	0.9	2.5	1.1	3.3

TABLE 3.—HEIGHT AND WEIGHT OF CONTROL BOYS (277)

Age, Years	Number of Cases	Initial Measurements		Gain per Year*	
		Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	9	....	16.3	....	....
1½	25	22.4	23.2	....	3.5
2	13	33.0	30.1	3.1	2.4
2½	4	35.2	28.0	2.2	3.9
3	20	35.4	30.0	2.3	3.6
3½	12	37.0	30.5	2.4	4.4
4	17	36.6	34.0	3.0	3.6
4½	5	41.0	36.4	2.7	2.1
5	20	40.4	37.6	2.3	4.1
5½	7	41.1	37.7	2.3	3.7
6	17	42.6	41.1	2.3	4.5
6½	4	42.2	42.0	1.9	6.4
7	19	45.0	44.0	1.5	4.8
7½	1	48.0	49.5	0.7	2.4
8	19	47.0	49.7	2.4	9.1
8½	4	48.0	49.0	0.8	4.5
9	16	49.5	54.5	1.3	4.4
9½	4	48.5	54.7	....	7.3
10	11	49.4	54.7	2.5	9.8
10½	4	53.5	74.0	1.9	1.4
11	12	52.6	63.6	0.9	....
11½	1	53.0	72.0	....	....
12	6	55.8	80.8	2.2	9.6
12½	..	....	....	2.2	....
13	20	57.0	77.6	....	....
13½	1	59.0	94.5	....	....
14	6	58.5	88.3	....	....

* This figure was computed by dividing the average gain, for a period equal in length to that of the tuberculous groups, by 12.

TABLE 4.—HEIGHT AND WEIGHT OF CONTROL GIRLS (337)

Age, Years	Number of Cases	Initial Measurements		Gain per Year*	
		Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	28	....	17.4	....	5.8
1½	13	....	29.0	....	0.0
2	12	32.8	25.5	1.9	4.1
2½	6	36.0	27.6	2.0	4.1
3	19	33.4	28.4	3.6	5.6
3½	12	35.2	29.0	3.1	4 5
4	21	36.6	33.4	2.5	3.0
4½	8	39.3	37.5	1.7	1.9
5	19	44.4	36.6	....	3.4
5½	8	42.0	37.7	0.9	3.5
6	21	42.3	39.4	1.5	3.8
6½	7	44.5	44.6	1.5	4.9
7	19	43.5	42.3	1.8	4.9
7½	8	44.0	47.2	1.7	3.8
8	20	45.5	47.7	2.4	5.6
8½	9	46.3	49.0	2.5	5.3
9	22	48.5	53.5	1.5	4.1
9½	..	....	....	....	....
10	29	50.0	63.0	2.7	3.1
10½	7	51.2	59.4	4.0	9.4
11	11	52.6	63.2	0.6	1.3
11½	1	56.0	70.5	....	....
12	16	....	....	....	....
12½	1	56.0	69.7	0.3	11.5
13	13	54.0	66.0	1.2	5.7
13½	1	54.0	62.7	....	....
14	6	56.6	89.4	....	....

* This figure was computed by dividing the average gain, for a period equal in length to that of the tuberculous groups, by 12.

TABLE 5.—WEIGHT AND HEIGHT OF BUREAU OF CHILDREN—BOYS

Age, Years	Measurements		Gain per Year (As in Table 3)	
	Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	29.3	21.8	3.3	4.7
1½	31.7	24.6	3.3	4.3
2	33.7	27.1	2.6	4.4
2½	35.3	29.5	2.4	4.4
3	37.1	32.2	2.0	4.0
3½	38.6	33.7	2.0	4.4
4	39.5	35.8	2.1	4.5
4½	40.5	38.4	2.1	4.6
5	41.6	41.1	2.1	4.3
5½	42.7	43.1	2.0	4.4
6	43.8	45.2	2.0	4.7
6½	44.7	47.1	2.0	5.6
7	45.7	49.1	1.9	5.3
7½	46.7	51.5	2.0	5.4
8	47.8	53.9	1.8	5.5
8½	48.7	56.5	2.0	5.7
9	49.7	59.2	1.7	5.3
9½	50.7	62.2	1.6	5.4
10	51.7	65.3	1.7	6.0
10½	52.3	67.7	1.9	7.0
11	53.3	70.2	1.8	7.0
11½	54.2	73.5	...	...
12	55.1	76.9	2.3	9.1
12½	56.1	80.8	2.3	9.1
13	57.2	84.8	2.6	11.1
13½	58.5	89.8	2.5	12.0
14	59.9	84.9	2.8	12.4

TABLE 6.—WEIGHT AND HEIGHT OF BUREAU OF CHILDREN—GIRLS

Age, Years	Measurements		Gain per Year (As in Table 3)	
	Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
1	28.8	20.7	3.3	3.8
1½	31.1	23.3	3.6	5.0
2	33.3	26.3	2.8	3.7
2½	34.8	28.2	2.3	4.1
3	36.7	30.5	2.1	3.8
3½	38.0	32.5	2.2	4.6
4	39.0	33.7	2.1	4.6
4½	40.1	36.7	2.2	4.4
5	41.3	39.7	2.1	5.7
5½	42.3	41.5	2.1	3.6
6	43.4	43.3	2.0	3.0
6½	44.4	45.4	2.0	5.6
7	45.5	47.5	1.9	4.8
7½	46.5	49.7	2.1	7.1
8	47.6	52.0	1.8	6.1
8½	48.5	54.5	1.9	6.2
9	49.4	57.1	1.9	5.7
9½	50.3	59.7	2.1	7.1
10	51.3	62.4	1.9	7.3
10½	52.3	65.6	2.3	8.2
11	53.4	68.8	2.3	10.0
11½	54.6	73.5	...	....
12	55.9	78.3	1.8	10.7
12½	57.0	83.5	1.8	9.4
13	58.2	88.7	1.4	8.6
13½	59.0	93.5	1.4	8.3
14	59.9	98.4	1.2	7.5

A study of the heights of boys shows that whereas seven of twenty-one tuberculous groups were normal in comparison with the Children's Bureau standard, the boys of the control series were normal in only five of twenty-five age periods. Furthermore, the number of individuals in these normal groups was 112 of 256 for tuberculous boys, or 43.75 per cent., but only 17 of 277 for control boys, or 6.13 per cent. In other words, the tuberculous males reached or surpassed the Bureau requirements for height seven times as often as those of the control series.

The figures for the girls of the tuberculous and control groups are less striking. The tuberculous girls were normal in six of twenty-three age periods while the control girls equalled the standard in only four of twenty-three age groups. The percentage of normality for these groups was 13.88 (35 of 252) for the tuberculous girls and 9.79 (33 of 337) for the controls. We note a definite though less marked superiority of the tuberculous females.

The tuberculous boys were normal in only two of twenty-six age periods, while the males of the control series were up to par in four of twenty-six groups. There were only four of 256, or 1.56 per cent., tuberculous boys who were of normal weight. However, twenty-four of 277 (8.66 per cent.) control boys reached the Bureau standard, almost five and one-half times as often as the members of the tuberculous series.

A consideration of these results shows that the type of boy in the tuberculous group was one whose height more frequently reached that of the Children's Bureau standard than did the control boy, but whose weight far less often was normal.

As for the weights of girls, it is apparent that three of twenty-six tuberculous groups were normal, while in the control group three of twenty-five were normal. With reference to the individual cases it is shown that 14.83 per cent. (50 of 337) of the control girls attained the Bureau standard, but only 1.98 per cent. (5 of 252) of the tuberculous series attained that standard. The control girls were thus strikingly more often of normal weight than the tuberculous girls (7 : 1).

Here, as with the boys, the tuberculous girl is comparatively taller but of a lesser weight than her sister of the control series. Both are much below the Children's Bureau standard.

With reference to height, the tuberculous boys were normal in 150 of 256 cases, or 58.59 per cent., as compared with 43.75 per cent. (112 or 256) of the averages for age groups. The control males were equal to the bureau norm in 88 or 243 observations, or 36.21 per cent., a figure which is much higher than the 6.13 per cent. (17 of 277) for height.

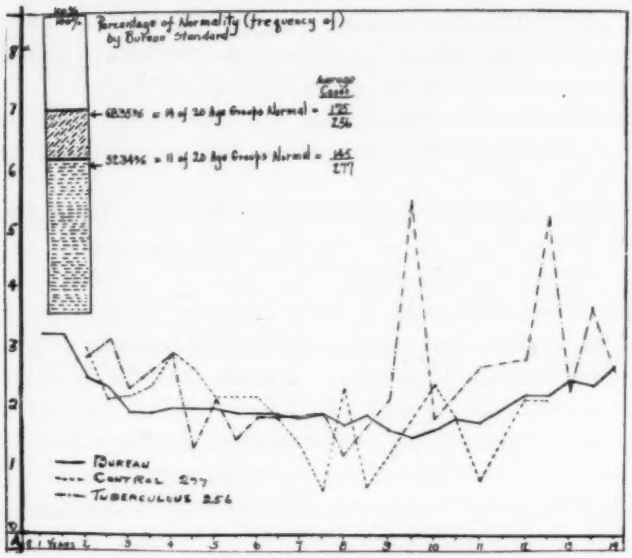
Percentage of Normality (frequency of) by Bureau Standard
Average Gains
68.35% = 13 of 20 Age Groups Normal = 1.75 / 2.54
55.34% = 11 of 20 Age Groups Normal = 1.45 / 2.77
BUREAU
CONTROL 2.77
TUBERCULOUS 2.56
Age in Years

Fig. 1.—Average gain in inches of different groups of boys per year.

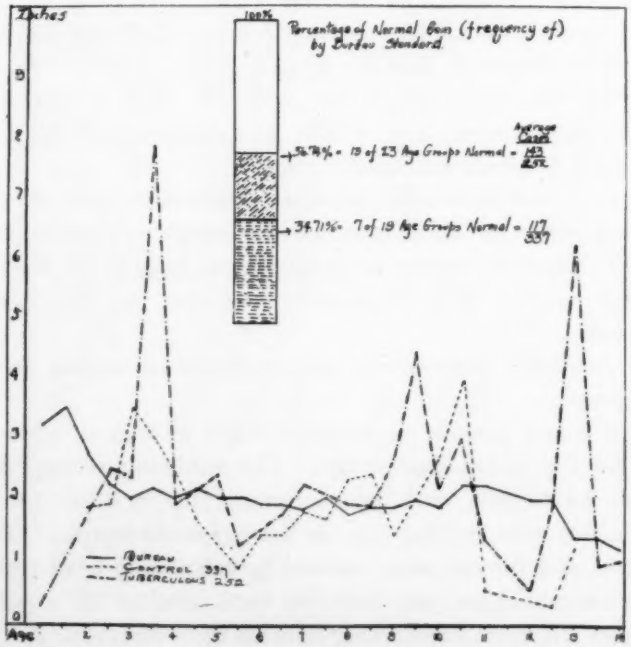
Inches
Percentage of Normal Gain (frequency of) by Bureau Standard
Average Gains
56.78% = 13 of 23 Age Groups Normal = 1.43 / 2.52
34.71% = 7 of 19 Age Groups Normal = 1.17 / 3.37
BUREAU
CONTROL 3.37
TUBERCULOUS 2.52
Age

Fig. 2.—Average gain in inches of different groups of girls per year.

The weight of the tuberculous boys reached the standard in 11.32 per cent. (29 of 256) as compared with the 1.56 per cent. (4 of 256) of the averages for age groups. Similarly, the percentage of normality is higher for control boys, 25.52 per cent. (74 of 277).

However, the fact remains that the tuberculous boys were more often of normal height than the controls, but were not as frequently of the normal weight.

As for the height and weight of girls, the tuberculous girls were of normal height in 58.29 per cent. (147 of 252) in contrast to 33.22 per cent. (98 of 295) for the controls.

With reference to weight, however the tuberculous girls were normal in only 1.98 per cent., and the controls in 14.83 per cent. Our individual observations, instead of average, indicate that the tuberculous girls were normal in 26.19 per cent. of the cases (66 of 252) while the control girls were slightly below this percentage, 23.69 per cent. (80 of 336 observations). The explanation for the difference lies in the fact that a large number of the individuals in the subnormal average age groups were equal to the Bureau norm.

The heights and weights of the boys and girls were added to give a composite. The tuberculous children were up to the standard in height in 58.47 per cent. of the cases (297 of 508), and the controls in 35.58 per cent. (186 of 538), so that the tuberculous child in individual instances attained the Children's Bureau norm, 22.89 per cent. (58.47—35.58), more frequently than the control.

However, the tuberculous child was less often of normal weight, 18.71 per cent. normality (95 of 508), as contrasted with 25.05 per cent. (154 of 615) for the control individual.

My conclusion as to tuberculosis in childhood is that given a group of children below the norm in height and weight, the tuberculous child will more frequently appear to be of normal height, but far less often of normal weight. The tuberculous individual is often seen as the lanky child.

This concludes the observations on the initial heights and weights of the groups.

Tables 1 and 2 show the average length of time of observation in months for the various age groups. The minimum average period for boys was 14 months, and the maximum 51.5 months: for girls the minimum was nine months, and the maximum 69 months. The curves for control and Bureau were secured by calculating what these groups would have gained per year had they been watched for a period equal to that over which the tuberculous children were observed. For example: The height of a Bureau boy of three years is 37.1 inches. If this child had been observed for 50 months, as was the tuberculous boy of the

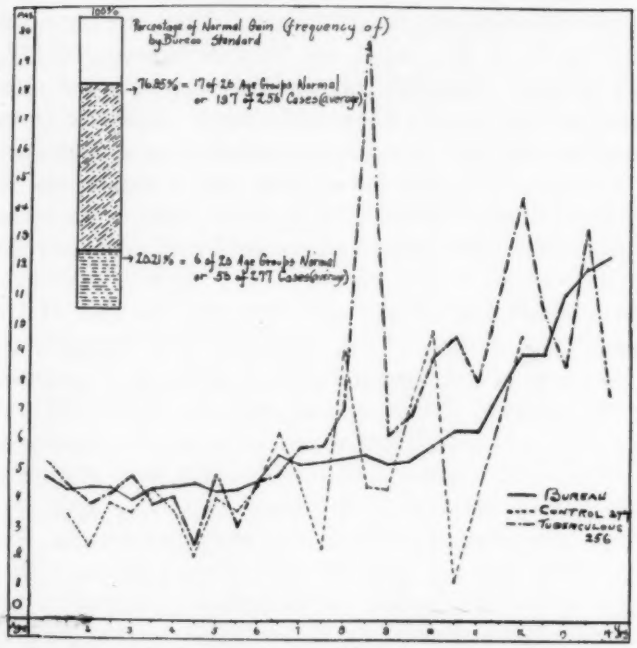
Percentage of Normal Gain (Frequency of) by Bureau Standard
76.85% = 17 of 26 Age Groups Normal or 197 of 256 Cases (Average)
20.21% = 6 of 26 Age Groups Normal or 55 of 277 Cases (Average)
BUREAU
CONTROL 277
TUBERCULOUS 256

Fig. 3.—Average gain in pounds of different groups of boys per year.

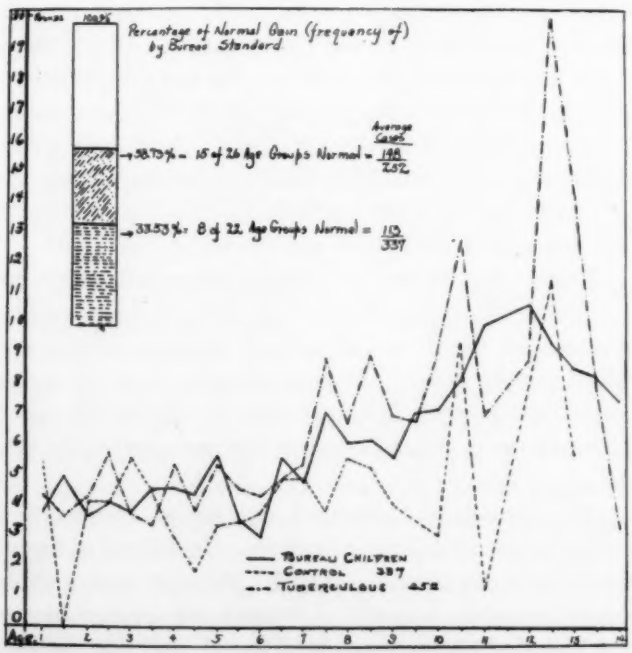
Percentage of Normal Gain (Frequency of) by Bureau Standard
38.73% = 15 of 26 Age Groups Normal = 198/252
33.33% = 8 of 22 Age Groups Normal = 113/337
BUREAU CHILDREN
CONTROL 337
TUBERCULOUS 452

Fig. 4.—Average gain in pounds of different groups of girls per year.

corresponding group, his height would have been 45.7 inches, a gain of 8.6 inches in this period of 4 2/12 years. His gain for a year would have been 12/50 of 8.6 inches, or 2.0 plus inches. In Chart 1 this figure was plotted. Similarly the gain was secured for other periods of the Bureau and the control series and plotted. With the Bureau curve as the standard the relative average gains of the tuberculous and control groups were studied. The tuberculous curve stands well above the control and the Bureau lines. The essential point to me appears to be how many of the tuberculous and control groups attained the Bureau norm.

Chart 1 shows the average gain in inches per year of boys in the three series. In fourteen of twenty age periods, the tuberculous boys crosses the curve of the Bureau standard, while eleven of twenty of the controls did likewise. These groups represent 175 of 256 tuberculous males, or 68.35 per cent. gained as well or better than the Bureau standard; while a smaller number, 145 of 277 control boys, representing 52.34 per cent. reached or passed the norm. Thus it is apparent that the tuberculous boys attained the standard more often than the control boys.

Chart 2 indicates the comparative average gain per year in height of the girls of the three series. In thirteen of twenty-three age periods the tuberculous girls attained the norm (56.00 per cent.) while in seven of nineteen (37.00 per cent.) the control females were up to par. More than half of the tuberculous girls equalled or surpassed the Bureau standard, 143 of 252 or 56.74 per cent. In contrast, the control girls were normal in height gain in 34.71 per cent., 117 of 337 cases.

Thus the tuberculous girls, even as the boys, equalled the Bureau gain more frequently than the control. It is interesting to note that in the very much undernourished control and tuberculous series the boys gained uniformly more normally than the corresponding girls. This fact is in accordance with the common observation in normal children, that on the whole boys gain better and are taller than girls.

Chart 3 demonstrates the remarkable better gain of the tuberculous boys over the control series. In 76.95 per cent. of the cases (197 of 256) and in seventeen of twenty-six age groups the tuberculous males attained the standard weight gain. The control males were up to par in only six of twenty age groups, 20.21 per cent. for 50 of 277 cases. As for degree of pound gain, the tuberculous line rises markedly after the age of seven.

The tuberculous girls (Chart 4) did not as frequently attain the Bureau weight gain as did their brothers. In fifteen of twenty-six age groups there were 148 normals of 252 (58.73 per cent.) while the controls crossed the norm in eight of twenty-two age groups for a total of 113 of 337 observations, or 33.53 per cent. The tuberculous girl

gained in weight normally in 25.20 per cent. more instances. Here likewise the degree of weight gain is very marked from the age of seven on, in the tuberculous series.

The superiority in gain in height and weight per year of the tuberculous series has been plotted in Chart 5. The frequency of normal gain in height per year of the tuberculous child was 62.59 per cent. (318 and 508 observations) as contrasted with 42.67 per cent. (262 of 614 observations) for the control child. In 67.91 per cent. of the cases (345 of 508) the tuberculous child attained the Children's Bureau standard, while the control child did as well in but 27.52 per cent. (169 of 614).

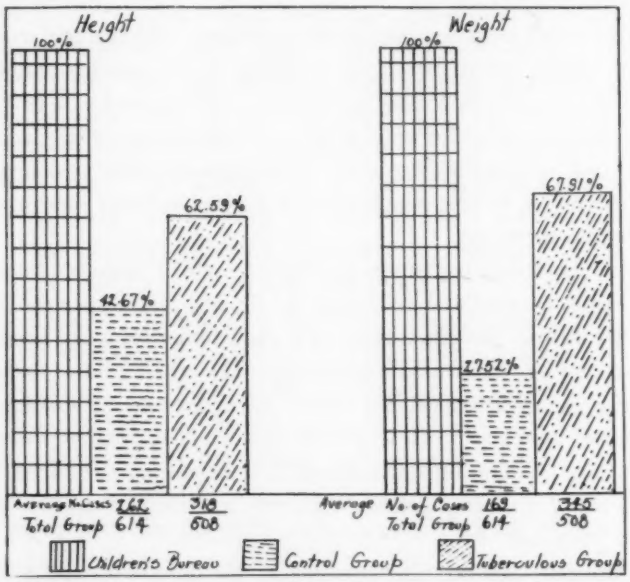

Fig. 5.—Composite of boys and girls. The percentage of normal gain per year of observation in comparison with the children's bureau standard.

CONCLUSIONS

1. Using height and weight as the basis of comparison, tuberculous children more frequently attained the normal standard of gain than those of the control series.

2. Neither the entire tuberculous nor control series reached the Children's Bureau standard, represented as 100 per cent., although the tuberculous group more frequently approached it.

3. The superiority of the tuberculous over the control series in gain, I believe to be dependent on the improvement in sanitation, food, and the removal of defects.

4. *The child with hilous or tracheobronchial gland tuberculosis in a group of children under-height and under-weight, when properly treated, will more often be the one to attain the normal gain.* This observation, according to my experience, is equally applicable to cases seen in private practice.

PROGRESS IN PEDIATRICS

RÉSUMÉ ON THE CIRCULATORY SYSTEM LITERATURE OF 1920*

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THE HEART

The *electrocardiogram* during the past year has become more extensively used, and more carefully studied. There are many points on which observers disagree, but certain fixed conclusions stand out clearly and its value in aiding diagnosis is undisputed. It is interesting to learn that in all of Paris Ribierre [1] knows of only two hospitals equipped for electrocardiography. To remedy this he suggests that medical societies in the smaller cities should acquire equipment to be loaned to the members.

Mann [2] describes his monocardiogram, which is an attempt to combine the graphs obtained in Leads I, II and III into a single curve. It is based on the fact that Lead II equals Lead III plus Lead I or the algebraic sum of the ordinates in Leads III and I at the same instant. By finding the location of the center of negativity at subsequent time intervals and connecting these points a curve is formed representing the cardiac cycle. This monocardiogram is a fusion of the three leads into a single curve by an algebraic reversal of the process by which the leads are obtained.

Hamburger [3] studies the changes in the P wave, which represents *the auricular complex*. The physiologic P complex is an upright wave which results from auricular systole. It expresses the origin of the heart beat at the normal site of the impulse formation, and the passage of the wave through the auricular tissue in definite directions. Alterations in the height, direction and configuration are caused by abnormal auricular conditions. Twenty per cent. of the 300 electrocardiograph curves at the Michael Reese Hospital (Chicago) show an inverted P wave, usually in Lead III. The P wave may be inverted by vagus stimulation, by the impulse originating in the lower portion of the auricle, by force expiration, and by digitalis. The P wave may be

* Received for publication, Jan. 25, 1921.

1. Ribierre, P.: Méd. mod. **1**:325 (March) 1920.
2. Mann, H.: Arch. Int. Med. **25**:283 (March) 1920.
3. Hamburger, W. W.: Arch. Int. Med. **26**:232 (Aug.) 1920.

persistently inverted, or an inverted P wave may sometimes become upright with atropin. In *auricular paroxysmal tachycardia* the P wave is apt to be inverted. White [4] analyzes a series of electrocardiograms in which the P wave is normal and the origin of the beat is not ectopic or, at least, if it be ectopic, it originates close to the normal pacemaker in the sinoauricular node. This is Galli's nomotopic (nomos = custom, topos = place) type of paroxymal tachycardia, and it has a better prognosis than the usual paroxysmal tachycardia. When the pacemaker moves toward the ventricle there is a tendency toward shortening the P-R interval unless there is a simultaneous vagal stimulation, and the P wave is flattened and finally inverted. White includes in the series two electrocardiograms of sino-auricular heart block not due to digitalis. There are few electrocardiograms of similar conditions in the published records.

Lewis [5] and others, in a series of papers on *flutter and fibrillation*, have brought out a new conception of the arrangement of the muscles in the auricle, and of the path of the excitation wave through these muscles. Auricular flutter shows in the electrocardiogram rapid, contiguous waves uniform in extent and outline. The excitation wave passes through the auricle in a regular sequence from cycle to cycle, without any moment when all the muscle fibers are at rest, as they are in true diastole. The rates of conduction are slower than the rate with normal rhythm, and the direction of the wave of excitation after flutter is established has no relation to the point at which stimulation is applied. When the course is distinctly displayed it extends around the mouths of both the superior and inferior vena cava veins, probably in "a continuous circus movement in a natural ring of muscles existing in the intact heart." It may run either clockwise or counterclockwise. The auricular appendages and the sleeves of muscle on the great veins are supplied by excitation waves centrifugally from the body of the auricle—waves thrown off from the central path as the central wave revolves, so that the direction of the wave in these outlying paths is constant, and depends on the direction of movement in the central path. The basis of flutter is a "circus movement following a central path in the auricles." After long continued experimental flutter in dogs, there are slight variations in the path which Lewis classes as impure flutter. Experimentally, the processes of auricular flutter, impure flutter and auricular fibrillation are linked as parts of a whole complex.

4. White, P. D.: Arch. Int. Med. **25**:420 (April) 1920.
5. Lewis, Th.: Heart **7**:127, 131, 247, 293 (April, Aug.) 1920.

Eyster and Meek [6] have a casette changing mechanism allowing two roentgenograms of the human heart to be taken in the same cardiac cycle. With a simultaneously recording electrocardiograph, they get curves from which the exact incidence of the exposures in the cardiac cycle may be determined. No single border of the heart may be used as an accurate index of contraction, the movement of the entire outline must be considered. The associated electrocardiograms and the roentgenograms show that most of the filling of the ventricle occurs shortly after the ventricle relaxes, and it is almost completed before the auricle contracts. The auricular rôle in adding blood to the ventricles is slight.

Bazett [7] finds that the duration of the *ventricular complex* in the electrocardiogram of the normal heart is a function of the pulse rate and may be determined by the formula Systole $= \sqrt{}$ cycle where $K = 0.37$ for males, and 0.40 for females. Exercise causes an increase in K, and an augmented vagal inhibition lowers K. Wiggers and Katz,[8] determining the cycle length and the duration of systole in a slow vagal heart beat, construct a probable volume curve for each animal and devise a plot of the theoretic relation between the length of systole and the cycle of any heart rate where the heart beats according to a uniform law. The accelerator nerves act on the ventricular muscles causing a reduction of the contraction period.

Brugsch and Blumenfeld [9] record heart sounds on phonocardiograms, controlled by electrocardiograms made at the same time in order to study the proportionate duration of ventricular systole expressed as a per cent. of the complete cardiac cycle. The variation in the different individuals and in the sexes is slight. The percentage length of ventricular systole increases with age. From 25 to 70 years of age the value is from 33.5 to 37.5 per cent. In aortic and mitral insufficiency the proportional duration of systole increases with the severity of the disease.

Fahr [10] follows the *wave of excitation through the ventricles* by following the change in direction of the vector or potential difference through a heart cycle, and then applying what we know of the structure of the His-Tawara-Purkinje system; of the rates of conduction through ordinary muscle; and of the time of onset of the first heart sound and the ventricle pressure wave. The method of the equilateral triangle is accurate within $\pm$ 10 degrees, and is used for determining the frontal plane projection of the direction of potential difference in the human

6. Eyster, J. E. A., and Meek, W. J.: Am. J. Roentgenol. **7**:471 (Oct.) 1920.
7. Bazett, H. C.: Heart **7**:353 (Aug.) 1920.
8. Wiggers, C. J., and Katz, L. N.: Am. J. Physiol. **53**:49 (Aug.) 1920.
9. Brugsch and Blumenfeld: Berl. klin. Wchnschr. **56**: (Oct. 6) 1920.
10. Fahr, G.: Arch. Int. Med. **25**:146 (Feb.) 1920.

ventricle, and the manifest value. The ventricular wave of excitation begins in the subendocardial layer of the Purkinje network, developing a little earlier in the right ventricle, then spreads to the apical layers causing the Q wave. The excitation process extends to the basal arborizations of the Purkinje network, giving the anacrotic limb of R. In the meantime, the process has spread from the apical network to the apical heart muscle, and when the peak of the R wave is reached, the negativity of the apical muscle fibers is great enough to neutralize the preponderance of basal Purkinje arborizations and to cause the catacrotic limb of R and the S peak. At the base of the heart, the excitation process is extending from the basal Purkinje network to the muscles, and their negativity increases until the effect of the apical fibers is reduced and the S wave is reduced. The negative process at the apex dies first, leaving a negative preponderance at the base, causing the T wave. In hearts with left sided enlargement, the electrocardiograph changes are due to an increase in the length of the conducting path in the right ventricle, the right side receiving its negativity first while in right sided enlargement the preponderance of negativity is on the left side. In otherwise normal hearts the form of the Q R S group is dependent on the relative weights of the two ventricles. The interpretation of the ventricular complex is not always clear. Pardee [11] says that hypertrophy of the left ventricle is expected to show a tall R wave in Lead I, with an absent S; and a small or absent R with a deep S wave in Lead III. Hypertrophy of the right ventricle is expected to show a relatively small R with a deep S Lead I and an absent S and a tall R wave in Lead III. The simplest most accurate formula for the L/R ratio is that of Lewis $(R_1 + S_3)—(S_1 + R_3)$. An increased L/R ratio gives R_3 relatively small and R_1 and S_1 large. This is a left predominance. A decreased L/R gives R_1 small and S_1 and R_3 relatively large.

Willius [12] studies changes in the form of the *initial ventricular complex* which is represented by the Q R S waves of the electrocardiogram. Willius divides a series of 747 cases into two groups—Group 1, including 14 per cent., shows a notched Q R S complex, while Group 2 shows slurred or thickened Q R S. In the first group more than half of the cases had predominance of the left ventricle, and the etiologic factors in order of their frequency are: (1) degenerative processes; (2) infections; (3) local nutritional disturbances, and (4) congenital heart disease. In the second group, the etiologic factors are infection and degeneration. Notched and thickened Q R S complex is frequently found in undoubted cases of myocarditis and probably means local disorders in the ventricular myocardium. Smith [13] gives

11. Pardee, H. E. B.: Arch. Int. Med. **25**:683 (May) 1920.
12. Willius, F. A.: Arch. Int. Med. **25**:550 (May) 1920.
13. Smith, F. M.: Arch. Int. Med. **26**:205 (Aug.) 1920.

a good review of the literature on atypical Q R S waves, and in experimental study in dogs of variations in the Q R S waves to determine the effect of lesions in the main branches of the A-V bundle, and of the arborizations of the bundle. His most interesting finding is that arborization block does not explain abnormal Q R S waves. Fatigue alone does not explain abnormal Q R S waves.

Fahr [10] explains that the *diagnosis of right and left bundle branch* lesion is probably made wrong, that in left bundle branch lesions R should be high in Lead I and S deep in Lead III and in right bundle branch lesion S should be deep in Lead I and R high in Lead III. Wilson and Herrmann [14] disagree with this view of Fahr concerning a mistake in the interpretation of the meaning of right and left bundle lesions. They say that in man the excitation wave passes down the Purkinje network of the septum, then upward through the network of the free walls inactivating the muscle in the same order. A lengthened P-R interval means delayed conduction through the A-V node or the main stem of the bundle of His. A lengthened Q R S depends on the thickness of the septum and is the most reliable sign of bundle branch block, the increase in amplitude having less value. The notching of Q R S complex is not uncommon in a normal electrocardiogram. The diagnosis of arborization block is on very shaky ground. The cause of the exaggerated T is not well understood. T deflection is produced by deactivation of the ventricular bundle.

Pardee [15] gives the electrocardiogram of a woman believed to have a lesion in the left ventricle, due to *occlusion of a coronary artery,* who died of an attack of angina pectoris two years later. A day or two after the obstruction the electrocardiogram shows the Q R S group usually notched in two leads, with prominence of the left ventricle. The T wave does not start from the zero level in Leads I and III. It quickly turns away, from the starting point in a sharp curve, without the short straight stretch preceding the peak of the T wave which is so evident in normal records. There are five other records in the literature that are similar. Klewitz [16] finds a negative T peak only in cases of organic heart disease, especially with myocardial lesions, but its absence does not rule out heart disease. Herrmann [17] adds electrocardiograph findings in six more cases of coronary thrombosis with ventricular tachycardia, three of which were proven at necropsy to be cases of coronary thrombosis. On of these is the first clinical case recorded which substantiates the experimental work of Lewis.

14. Wilson, F. N., and Herrmann, G. R.: Arch. Int. Med. **26**:153 (Aug.) 1920.
15. Pardee, H. E. P.: Arch. Int. Med. **26**:244 (Aug.) 1920.
16. Klewitz, F.: Deutsch. Arch. f. klin. Med. **129**:41 (April 29) 1919.
17. Herrmann, G. R.: J. Missouri M. A. **17**:406 (Oct.) 1920.

All six patients clinically had severe anginal attacks and pulse rates from 170 to 250 per minute and the electrocardiograms are those of paroxysmal ventricular tachycardia.

Bishop [18] offers two cases showing early signs of *ventricular fibrillation* as periods of ventricular tachycardia which he considers a serious factor in sudden death from heart failure, due either to fatigue of the ventricular muscles from the rapid rate of contraction or to fibrillation of the ventricle.

McCulloch,[19] studying *electrocardiograms from athreptic infants,* finds alterations of the cardiac mechanism in two cases; in the form of a delayed auriculoventricular conduction and changes in the Q R S complex. These alterations disappear as the child improves.

Jones [20] made *electrocardiographs after the cessation of spontaneous respiration* in a case of cerebral hemorrhage where the heart beat for fifteen hours after normal respiration ceased, and intratracheal insufflation was used; after twelve hours under artificial respiration the heart has normal rhythm, with some lengthening of the ventricular complex that may at times exceed 0.5 second in duration.

Klewitz [21] finds that the *electrocardiogram in persons asleep* differs materially from the electrocardiogram of the same person awake. During sleep the heart cycle is lengthened, the ventricular systole is proportionately prolonged. In different forms of heart disease no regularity can be detected in the sleep change, one case of bradycardia showing a shortened heart cycle.

Josue [22] uses *auscultation of the venous pulse* to obtain information otherwise obtainable only by cardiograph methods. With the patient flat, and the head low, the stethoscope is applied between the sternal and clavicular attachments of the sternocleidomastoid muscle. The stethoscope should almost parallel the neck axis, slanting downward, backward and inward, pointing to the mediastinum. It is applied just back of and as close to the clavicle as possible, with a minimal amount of pressure. The sounds resemble the peaks in the electrocardiogram, and may be interpreted by the radial pulse relationship.

VAGUS EXCITABILITY OF THE HEART

Vagus control and the cardiac reflexes have been the subject of numerous experiments. Laslett [23] examined 108 persons in whom there was a striking reaction to vagus pressure, the mechanism being determined by slowing of the venous or radial curves. There is little

18. Bishop, L. F.: Ann. Med. **1**:58 (April) 1920.
19. McCulloch, H.: Am. J. Dis. Child. **20**:486 (Dec.) 1920.
20. Jones, H. W.: Lancet **2**:501 (Sept. 4) 1920.
21. Klewitz, F.: Deutsch. Arch. f. klin. Med. **130**:212 (Sept. 26) 1919.
22. Josue, O.: Bull. méd. Par. **34**:391 (April 28) 1920.
23. Lazlett, E. E.: Heart **7**:347 (Aug.) 1920.

practical difference in the predominance of right or left nerve. The right nerve has slight predominant control over the auricular rate, but the left also retards the ventricular rate. The ratio is R: L = 68: 55. The left vagus is predominant in control of the auriculoventricular conduction, the ratio being R: L = 15: 23.

Kleemann [24] used Czermak's test pressure on the vagus in 127 patients. Tests on the right vagus showed no effect in 80 out of 150 tests, and on the left vagus no effect in 90 out of 149 tests. Marked modification is only evident in cases of heart disease, but the intensity of the response is not always proportional to the severity of the disease, and a positive response does not always occur with heart disease. When there is partial heart block the response is aggravated. This brings the vagus pressure experiment into the medicolegal realm. Von Teubern [25] points out that in persons with a strongly positive vagus response compression of the throat for a very short space of time, as in attempts to strangle, or choking, may be sufficient to produce immediate arrest of the heart action.

Valsalva's experiment, i.e., forced expiration after deep inspiration with the nose, mouth and glottis closed, according to Maudru [26] gives a chance for better exploration of the heart, because the lungs are still, the heart rate is slower, so that murmurs are more easily studied, and the more intimate contact of the layers of the pericardium may bring out unexpected friction sounds. Dawson and Hodges [27] find that with the Valsalva experiment systolic blood pressure rises rapidly, at first, and this is followed by a marked fall. When the experiment is prolonged the intensity of efforts decreases and with this the changes in the heart rate are less marked. When the Valsalva experiment is interrupted by a series of single gasps, the systolic blood pressure rises from 10 to 20 mm. above the normal. Lifting and "exercises of strain" have this same effect.

The oculocardiac reflex is used for determining the vagus-sympathetic balance. Jenny [28] applied the compression of eyeball test to 250 children whose ages varied from 3 months to 15 years. In only two cases was there a disagreeable effect and the pulse was slowed in all but 4.4 per cent. which included five cases of tuberculous meningitis and two of diphtheria. The oculocardiac reflex is a physiologic reflex which ranks in value with the knee jerk. It tests the vegetative system, and is especially valuable for testing the heart as a control in digitalis treatment. Lesne and Binet [29] induce more vigorous responses in the

24. Kleemann: Deutsch. Arch. f. klin. Med. **130**:221 (Sept. 26) 1919.
25. Von Teubern: Deutsch. med. Wchnschr. **46**:322 (March 18) 1920.
26. Maudru: Paris méd. **10**:504 (July 19) 1920.
27. Dawson, P. M., and Hodges, P. C.: Am. J. Physiol. **50**:481 (Jan.) 1920.
28. Jenny, E.: Arch. f. Kinderh. **68**:64 (Aug. 31) 1920.
29. Lesné, E., and Binet, L.: Arch. de méd. d. enf. **23**:69 (Feb.) 1920.

oculocardiac reflex for testing vagus excitability in children than adults. In infants 2 weeks old, pressure on both eyeballs causes the pulse rate to drop from 120 to 130 down to 90. It returns to normal at once. Guyot and Jeanneney [30] emphasize the need for using caution in applying the test, but find it very valuable in estimating the condition of a patient before operation. If the reaction is normal, the patient may be operated on; if it is inverted it means an upset between the vagus and the sympathetic control, with the sympathetic predominating. This is the condition found in some forms of shock. When the reflex is abolished, there is insufficiency of the medullary centers, and surgery must be cautious.

Prevel [31] speaks of the *abdominocardiac reflex*. This is the acceleration of the heart beat on changing from the reclining to the erect position. There is no alteration in the perfectly healthy person, nor in patients whose abdomen is supported by the abdominal band or by their hands. It is explained by traction from the sagging organs on the solar plexus.

ARRHYTHMIAS

Eyster and Swarthout [32] find that anything that interferes with the normal rate and coordination of the beats interferes with the mechanical efficiency of the heart. Extra systole, always a premature beat, and a deficient beat as compared with the normal, does not markedly reduce the mechanical efficiency; auricular fibrillation and auricular flutter cause a decrease of cardiac output of as much as 34.9 per cent. of the normal; auriculoventricular heart block decreases mechanical efficiency because of the slow ventricular rate. Barlaro [33] insists that treatment of arrhythmias must be based on the special form and the occasional cause such as gastro-intestinal involvement or impacted gallstones. Schrumpf [34] concludes that serious cases of tricuspid insufficiency are nearly always accompanied by complete arrhythmia.

Tachycardia, according to Wilson,[35] in a man who is up and around, is compensatory, pointing to a mild infection with the toxin acting directly on the cardiac vagus. Parxoysmal tachycardia is not incompatable with an active life. Chevallier [36] lists the most common methods of arresting the paroxysms by reflex action. These include swallowing a chunk of bread or drinking in big gulps, taking an emetic to induce

30. Guyot, J., and Jeanneney, G.: J. d. méd. de Bordeaux **91**:591 (Nov. 25) 1920.
31. Prével: Presse méd. **28**:235 (April 21) 1920.
32. Eyster, J. A. E., and Swarthout, E. C.: Arch. Int. Med. **25**:317 (March) 1920.
33. Barlaro, P. M.: Prensa Médica Argentina **6**:231 (Jan. 10) 1920.
34. Schrumpf, P.: Arch. d. mal. du cœur. **12**:529 (Dec.) 1919.
35. Wilson, R. M.: Lancet **1**:146 (Jan. 17) 1920.
36. Chevallier, C.: Bull. méd. Par. **34**:395 (April 28) 1920.

vomiting or tickling the pharynx with a feather, lying on the back and breathing with the chest kept full of air, pressure on the right vagus nerve in the neck, and taking every ten minutes a cachet of pituitary extract. Galli [37] reports an interesting case of paroxysmal tachycardia in which the attack can be produced at will by the giving of atropin.

Paroxysmal auricular fibrillation is found by Mason [38] in 7 per cent. of the cases of auricular fibrillation in the Johns Hopkins Hospital records. The electrocardiograms are given. Willius,[39] studying 500 cases of auricular fibrillation, finds that the mortality in these cases doubles and sometimes trebles that of heart disease not accompanied by this arrhythmia. Levy,[40] in a research on *ventricular fibrillation* in cats and dogs, finds that the ventricle of the cat tends to spontaneous recovery, and this tendency may be aided by massage of the heart and the use of artificial respiration. The time interval between the moment of syncope and the beginning of the massage is not the important factor in recovery. The limitation to the efficiency of massage is the extent of time during which the central nervous system may be deprived of blood (oxygen?) without injury. This is about five minutes as an outside limit in the human brain. The human heart reacts like the cat heart. Ventricular fibrillation in dogs does not tend to spontaneous recovery and is usually fatal, although D'Halluin [41] saved 65 per cent. of the dogs with ventricular fibrillation from exposure to electricity by the intravenous injection of solutions of potassium chlorid.

Esmein [42] collected the recent literature on the pathology of the bundle of His and its ramifications. Schrumpf [43] reports a case of interfering double sinus rhythm the records from which seem to sustain the view that there is a dual action of the right and left nodes of Keith.

Bishop [44] reports an interesting case of *symmetrically placed ventricular extra systoles* and a slow pulse which is very different from true bradycardia in which the pacemaker is slow, or from heart block where impulses do not reach the ventricle. In this case every other normal pulse beat is replaced by a premature contraction which is too feeble to be noticed in the pulse. There is no functional disorder of the mechanism, but each alternating beat is an ectopic beat originating in the ventricle, and is followed by a beat controlled by the normal

37. Galli, G.: Heart **7**:111 (April) 1920.
38. Mason, V. R.: Bull. Johns Hopkins Hosp. **31**:145 (May) 1920.
39. Willius, F. A.: Minnesota Med. **3**:356 (Aug.) 1920.
40. Levy, A. G.: Heart **7**:175 (April) 1920.
41. D'Halluin, M.: Jour. de Radiol. e. d'Electrolog. Paris **4**:254 (June) 1920; Abstr. J. A. M. A. **75**:1297 (Nov. 6) 1920.
42. Esmein, C.: Ann. de Méd. **6**:415 (Dec.) 1919.
43. Schrumpf, P.: Arch. d. mal. du cœur. **13**:168 (April) 1920.
44. Bishop, L. F.: Am. Med. **15**: (new series), April, 1920.

pacemaker. Willius [45] made a study of 277 patients with *bradycardia* and he finds that the causes may be chemical substances, myocardial disease, physiologic changes without cardiac degenerations, early and late asphyxia, and stimulation of the cardiac vagus which gives a temporary bradycardia.

Parkinson, Grosse and Gunson,[46] taking fifty consecutive cases of acute rheumatism, find the myocardium unaltered in only twelve cases. Forty-seven of the patients had a sinus arrhythmia after the attack and 14 per cent. had premature auricular contraction; 30 per cent. had acute heart block, with dropped beats in 8 per cent. Some degree of acute heart block indicating myocarditis is common in acute rheumatism. Goodall,[47] studying twenty cases of *heart block,* points out that rheumatic and syphilitic histories are relatively infrequent. Heart block is almost invariably associated with mitral regurgitation, and pain is a frequent symptom. There is a tendency to sudden death with gastric distention, and Stokes-Adams attacks occur when the heart block is complete. He speaks also of the possibility of transmission of the condition from mother to child.

Eckstein,[48] working on the frog's heart, confirms the view that the width of the impulse conducting tract between the auricle and ventricle does not modify the transmission of the impulse. Partial heart block is not due to the narrowing of the tract. The power of the impulse is dependent on the number of times the elements of the tract can repeat a given process in a given length of time. When two parts with varying lengths of the refractory phase meet there must be arrhythmia—which may be purely functional, due to injurious influences affecting the heart through the blood or nerves or originating within the heart itself. Cases of heart block are reported by Gallavardin and Dumas; [49] Friedlander and Isaacs,[50] whose case shows a dissecting aneurysm of the septum in a man, aged 36; Odriozola,[51] whose case with gumma of the septum shows complete heart block, and a pulse rate dropping to one beat per minute, probably the lowest bradycardia on record; and Pezzi, Donzelot and Yacoel,[52] whose case of complete heart block shows a pulse of auricular origin due to myocardial weakness, in the femoral vein.

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45. Willius, F. A.: Arch. Int. Med. **26**:630 (Nov.) 1920.
46. Parkinson, J.; Grosse, A. H., and Grunson, E. B.: Quart. J. Med. **13**:363 (July) 1920.
47. Goodall, J. S.: Lancet **1**:909 (April 24) 1920.
48. Eckstein, A.: Deutsch. Arch. f. klin. Med. **130**:95 (Aug. 15) 1919.
49. Gallavardin, L., and Dumas, A.: Arch. d. mal. du cœur. **13**:63 (Feb.) 1920.
50. Friedlander, A., and Isaacs, R.: J. A. M. A. **75**:1778 (Dec. 25) 1920.
51. Odriozola, E.: An de la Facultad de Med. de Lima **3**:5 (July-Aug.) 1920; abst. J. A. M. A. **75**:1601 (Dec. 4) 1920.
52. Pezzi, C.; Donzelot, E., and Yacoel, J.: Arch. d. mal. du cœur **13**:97 (March) 1920.

Heartblock in children is much more rare. Eyster and Middleton [53] could find only twenty cases of heart block in children in the literature. These were probably either congenital in origin or occurred during severe diphtheria. Their case is in a child, 2 years old, which has been under observation all her life. The lesion at present seems to be a well compensated mitral lesion, with a 2:1 auriculoventricular block, the ventricle rate being from fifty to sixty beats per minute. Carter and Howland [54] report a case of congenital *atrioventricular dissociation* with complete heart block in a child of 5. The auricular rate is 103 and the ventricular rate is 37. There are only seven other established cases of atrioventricular heart block in children. These cases are reviewed in the article.

THE SIZE AND CONTOUR OF THE HEART

Hoover [55] gives the fine distinctions of the lost art of percussion in locating the heart borders. Direct percussion is best for definitive percussion using the terminal phalanx of the extended middle finger, making the blow by firmly stroking, with the patient sitting because the heart is nearer the chest wall. The left cardiac border is located by percussing from the third intercostal space downward; then the upper border by percussing in a line midway between the sternum and the nipple line with the finger parallel to the ribs, downward each costal and intercostal space until there is the characteristic change in note and resistance. The right cardiac borders should be defined by percussing in each right intercostal space from the second to the fifth in succession. The location of the right margin requires all the niceties of technic. Not even the radiographic plate will give the information about flattening of the diaphragm with cardiac enlargement that may be obtained by studying the movement of the costal margins from the subcostal angle to the eighth costal cartilage. For this the patient should be recumbent. Normally, the two sides move symmetrically outward, but if the entire subcostal diaphragm is flattened, the costal margins on both sides from the angle to the eighth rib move less than the lower margins where the lateral phrenic leaves are attached. If the flattening is marked, the costal margins from the angle to the eighth rib move symmetrically inward, and the lower margins move outward, giving an inspiratory narrowing of the subcostal angle. This is a valuable clue to downward enlargement of the heart.

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53. Eyster, J. A. E., and Middleton, W. S.: Am. J. Dis. Child. **19**:131 (Feb.) 1920.

54. Carter, E. P., and Howland, J.: Bull. Johns Hopkins Hosp. **31**:351 (Oct.) 1920.

55. Hoover, F. C.: J. A. M. A. **75**:1626 (Dec. 11) 1920.

Martinet [56] uses the height, the diameter of the chest from axilla to axilla, the transverse diameter and the longest and shortest diameters of the heart. These furnish an index of the relative values of the heart and aorta for different builds, which is very instructive. Cánovas [57] uses a radiocardiometer to measure the ratio between the diameter of the heart and the widest diameter of the chest. Normally, this is almost constant, 25 per cent. of the chest diameter lying to the left of the heart, 42 per cent. being accupied by the heart, and 33 per cent. lying to the right of the heart. Cohn,[58] using the teleroentgen method on soldiers returned from severe active service, finds that their hearts are not larger than the normal.

IRRITABLE HEART AND EFFORT SYNDROME

Warfield and Smith [59] note that cases of *irritable heart* give histories dating back for years—and exercise under observation gives the surest method of separating the fit from the unfit. Galli [60] shows that cardiovascular disease is an inherited tendency, and he gives family trees for "hereditary myocardism." These subjects are unable to stand as much physical exercise as the normal. The heart and aorta are smaller than the average, and the blood pressure is lower. Kerley [61] places the intelligent child, who is unable to apply himself to either mental or physical work because he becomes easily fatigued, in the class of the effort syndrome in children. Schiff [62] reclassifies these children with constitutional inferiority of the circulatory system according to the stability of the vasomotor system. Stolte [63] calls attention to the children whose movements are slow, who do not join in games, who urinate little during the day, but at night when the circulation is relieved pass large amounts of urine. These children may have an indistinct first sound at the apex, or a murmur, which is due to atony of the heart muscles and which disappears as the child grows stronger. The effort syndrome is not heart disease. Meredith,[64] examining 2,000 young women, found 193 who had been told they had valvular lesions who gave no evidence of organic heart disease, but who had a functional disturbance that may be attributed to submyocardial development.

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56. Martinet, A.: Presse méd. **28**:302 (May 15) 1920.
57. Cánovas, B. N.: Rev. Española de med. y cir., Barcelona **2**:600 (Nov.) 1919.
58. Cohn, A. E.: Arch. Int. Med. **25**:499 (May) 1920.
59. Warfield, L. M., and Smith, F. M.: J. Lab. & Clin. M. **5**:75 (Nov.) 1919.
60. Galli, G.: Policlinico, Rome **27**:65 (Jan. 19) 1920.
61. Kerley, C. G.: Arch. Pediat. **37**:449 (Aug.) 1920.
62. Schiff, E.: Jahrb. f. Kinderh. **91**:217 (March) 1920.
63. Stolte, K.: Jahrb. f. Kinderh. **92**:1, 1920.
64. Meredith, F. L.: Boston M. & S. J. **181**:734 (Dec. 25) 1919.

ORGANIC HEART LESIONS

White [65] insists on positive evidence for the diagnosis of *heart disease* in young people. This positive evidence consists of definite cardiac enlargement, murmurs that are definitely those of organic valvular lesions, serious arrhythmia such as heart block or auricular fibrillation, congestion of the veins of the neck, enlargement of the liver, edema of the feet and legs, ascites, pulmonary edema and cyanosis. Hewlett [66] adds to this list cardiac infection and alterations in the form of the electrocardiogram. Gibre,[67] from the records on 10,000 recruits with doubtful heart conditions, selects the cases of aortic regurgitation and mitral stenosis. He finds that the most important etiologic factor between the ages of 18 and 41 is rheumatic fever. It is rather surprising to find the statement that there is no evidence here that syphilis, gonorrhea, scarlet fever, diphtheria, or pneumonia are causes of valvular heart lesions. Hall,[68] in 2,500 heart cases, analyzed the 40 per cent. who had valvular heart lesions. Half of these were due to want of tone and to minor causes, all curable with the proper environment.

Of the acute infectious diseases producing heart lesions diphtheria is one of the most important. McCulloch [69] gives the records of eighty cases of diphtheria in children, of whom nineteen showed cardiac disturbance. With lesions of the myocardium and the mechanism, the mortality is 100 per cent. Cardiac failure results the moment the cardiac reserve is used up. The occurrence of *postdiphtheritic myocarditis* is beyond our control, but the outcome may depend on protection given the heart during the disease and convalescence. Aviragnet and Lutembacher [70] report a case of postdiphtheritic derangement of conductivity in a boy, 10 years old. Minet and Legrand [71] report six cases of influenza with heart complications. The prognosis is grave for all organic forms and reserved in all types. Hamburger [72] adds six more cases of *postinfluenzal myocardial involvement*. These may be grouped as (1) fatal cases with acute parenchymatous degeneration and vacuolization of the myocardium, (2) acute nonfatal cases showing involvement of the auricle and the conducting system during the height of the infection and recovery in from two to six weeks, and (3) nonfatal chronic cases with involvement of the auricle and arrhythmia long after subsidence of the acute infection. Acute respiratory infections,

65. White, P. D.: J. A. M. A. **74**:580 (Feb. 28) 1920.
66. Hewlett, A. W.: Northwest Med. **19**:224 (Sept.) 1920.
67. Gibre, C. C.: Brit. M. J. **1**:730 (May 29) 1920.
68. Hall, G. R.: Lancet **1**:1218 (June 5) 1920.
69. McCulloch, H.: Am. J. Dis. Child. **20**:89 (Aug.) 1920.
70. Aviragnet and Lutembacher: Arch. d. mal. de cœur. **13**:197 (May) 1920.
71. Minet and Legrand: Paris méd. **10**:133 (Feb. 14) 1920.
72. Hamburger, W. W.: Am. J. M. Sc. **160**:479 (Oct.) 1920.

such as influenza and streptococcus lesions, seem to single out the auricle and the conduction pathways. Oliverira [73] finds that lung processes, especially fibrous degeneration of the lung, cause disturbances in the venous cardiovascular system which are slowly progressive and lead up to auricular fibrillation. This correlation between auricular fibrillation and the pulmonary condition is the basis for his term—*pulmonary heart.*

Williamson [74] injects a warm gelatin and agar mixture into the pericardial sac of fresh cadavers, to study *pericarditis with effusion.* The fluid accumulates first along the lower margin of the heart and about the apex; with larger effusions it collects next over the large vessels of the base. This collection is independent of position. In almost every case this pushes down the diaphragm and the left lobe of the liver. Even in large effusions the anterior surface of the heart may remain uncovered so that a pericardial friction rub may still be noted. Percussion dullness in the right fifth interspace, or rounding of the cardiohepatic angle is not demonstrable in experimental pericardial effusions. Holden [75] reports three cases with abdominal symptoms and laparotomy —in which the lesion was a pericarditis. Rehn [76] treats *adhesive pericarditis* by splitting the sternum, slitting the pericardium, and breaking up the adhesions. Four cases in children showed immediate improvement, and recovery from the operation, but two died soon afterward of tuberculosis elsewhere, one after eighteen months of influenza, and one after one year of acute rheumatism.

Dwyer [77] cites a case of *ulcerative endocarditis* in a child, 23 months old, due to the gonococcus. In over 600 cases of endocarditis in children there is only one mention of endocarditis due to the gonococcus. The most common agent is *Streptococcus viridans.* This was found in the case of Kretschmer and Conboy [78] in which both the aortic and mitral valves were perforated. Münzer [79] thinks that the presence of streptococci in the blood does not justify prognostic conclusions; that chills and splenic enlargement are more important for a bad prognosis. Baehr and Lande [80] believe that the glomerulonephritis which is the cause of death in 11.5 per cent. of their cases of subacute streptococcus endocarditis is initiated only during the bacteremia in the acute stage of

73. De Oliverira, O.: An. de la Facult. de Med. Montivideo **5**:297 (July-Aug.) 1920; Abstr. J. A. M. A. **75**:1808 (Dec. 25) 1920.
74. Williamson, C. S.: Arch. Int. Med. **25**:206 (Feb.) 1920.
75. Holden, W. B.: Northwest. Med. **19**:230 (Sept.) 1920.
76. Rehn, L.: Arch. f. Kinderh. **68**:179 (Oct. 16) 1920.
77. Dwyer, H. L.: J. A. M. A. **75**:1643 (Dec. 11) 1920.
78. Conboy, J. E., and Kretschmer, H. L.: J. A. M. A. **74**:154 (Jan. 17) 1920.
79. Münzer, E.: Zentralbl. f. inn. Med. **41**:282 (April 17) 1920.
80. Baehr, G., and Lande, H.: J. A. M. A. **75**:789 (Sept. 18) 1920.

endocarditis. Cotton [81] analyzes the clinical symptoms and complications in subacute endocarditis. Thomas and O'Hara [82] report a case of endocarditis of the tricuspid valve due to the pneumococcus, Type I. Hannemann [83] finds that the warty efflorescences in endocarditis are made up of valvular tissue and are not superficial deposits, the deposit of cells arising from the valvular tissue and not from leukocyte accumulations.

Kaufmann,[84] studying *large hearts* in soldiers, finds that most of these are not hypertrophied. The power of dilation is a compensating heart mechanism, but the larger the heart the less the relief from dilation. The amount of strain depends on the previous condition of the heart and not the severity of the exercise. Of one group of fifty men showing no disordered heart action after two years at the front, only eight had had acute infections after the age of 14; while in another group of fifty men who were returned with severe heart symptoms after five months at the front, thirty-seven had had acute infections after the age of 14. Acute dilatation of the heart during or following surgical operation, in nine cases reported by Levine,[85] showed abnormal mechanism of the heart beat, with the changes in the auricular beat. Esmein [86] finds that the auriculoventricular valves are more apt to become insufficient with dilatation which really lightens the work of the heart. Wilson [87] says that every case of cardiac decompensation without valvular lesions must be considered a case of chronic myocarditis. Leon [88] and Morquis [89] report cases occurring in children in which a toxic action in the myocardium and compensatory dilatation caused a *gallop sound* which directed the attention to an acute nephritis. Lian [90] considers the *third heart sound* physiologic, and Pezzi [91] opposes the view that a double crural sound is a symptom of aortic insufficiency. It is of mixed arterial and venous origin, is never present with compensated aortic insufficiency, and it occurs only with myocardial weakness.

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81. Cotton, T. F.: Brit. M. J. **2**:851 (Dec. 4) 1920.
82. Thomas, H. M., Jr., and O'Hara, D.: Bull. Johns Hopkins Hosp. **31**: 417 (Nov.) 1920.
83. Hannemann, E.: Virchows Arch. f. path. Anat. **226**: Supp. 138-149, 1919; Abstr. Bacteriol. **3**: (Dec.) 1919.
84. Kaufmann, R.: Wien. Arch. f. inn. Med. **1**:211 (June) 1920.
85. Levine, S. A.: J. A. M. A. **75**:795 (Sept. 18) 1920.
86. Esmein, C.: Médecine, Paris **1**:336 (March) 1920.
87. Wilson, F. N.: Mississippi State M. A. J. **17**:403 (Oct.) 1920.
88. Ponce de Leon: Arch. Latino-Amer. de Pediat., Buenos Aires **14**:266 (May-June) 1920.
89. Morquis, L.: Arch. Latino-Amer. de Pediat., Buenos Aires **13**:424 (Oct.) 1919.
90. Lian C.: Médecine, Paris **1**:333 (March) 1920.
91. Pezzi, C.: Arch. d. mal du cœur **12**: (Dec.) 1919.

Rosenbaum,[92] studying *cardiac complications in scarlet fever,* finds myocarditis common in the latter part of the acute stage and in convalescence, but a pre-existing old heart lesion does not mean a bad prognosis in scarlet fever. Whitman and Eastlake [93] trace all stages from muscle cell to Aschoff cells in rheumatic myocarditis. The myocardial rheumatic nodule, or Aschoff body, is made up of large elements arranged radially about a center. The cells are similar to epithelioid cells, but larger like the giant cells in Hodgkin's disease. They are round, fusiform or oval, often multinuclear. The nodules lie close to small blood vessels and are surrounded by a zone containing polymorphonuclear leukocytes, plasma cells and lymphocytes.

Norton [94] reports a case of myxoma of the heart in the hope of finding a symptom complex for *cardiac tumors.* Rutherford [95] reports a case of *cardiac angina in a child,* with a review of the literature of angina pectoris and degenerations in the aorta in children. Lutembacher [96] cites three cases of aneurysm of the left ventricle in adults. Reid [97] reports an experimental study on dogs with experimental *arteriovenous fistulae.* There is a marked tendency for such experimental fistulae to close.

WOUNDS OF THE HEART

Mocquot and Constantini [98] report eleven cases in which the heart wound in the ventricle is plugged with a clot, and may heal spontaneously, although if this clot becomes destroyed, there may be secondary hemorrhage and delayed symptoms. Collins [99] sutured the heart in a case of gunshot wound of the left ventricle. The man lived thirteen days, and died of the effect of exposure due to an attempted escape from the hospital. At necropsy the heart and pericardium were completely healed. Proust [100] records the results of a re-examination ten years after suture of the heart, in which the heart is apparently normal. In the discussion of this paper Josue gives the findings in ten cases in which there were operations on the heart after war wounds.

CONGENITAL ANOMALIES OF HEART AND CIRCULATION

Vaquez and Donzelot [101] distinguish between *dextrocardia* and

92. Rosenbaum, H. A.: Arch. Int. Med. **26**:424 (Oct.) 1920.
93. Whitman, R. C., and Eastlake, A. C.: Arch. Int. Med. **26**:601 (Nov.) 1920.
94. Norton, W. H.: Am. J. M. Sc. **158**:689 (Nov.) 1919.
95. Rutherford, W. J.: Brit. J. Child. Dis. **17**:22 (Jan.-March) 1920.
96. Lutembacher, R.: Arch. d. mal. du cœur **13**:49 (Feb.) 1920.
97. Reid, M. R.: Bull. Johns Hopkins Hosp. **31**:43 (Feb.) 1920.
98. Mocquot, P., and Constantini: Rev. de chir. **39**:257, 1920.
99. Collins, A. W.: J. A. M. A. **75**:1271 (Nov. 6) 1920.
100. Proust: Bull. et mém. Soc. méd. d. hôp. de Par. **44**:1035 (July 16) 1920.
101. Vaquez, H., and Donzelot, E.: Presse méd. **28**:41 (Jan. 17) 1920.

dextroversion; one, the mirror aspect, being merely a mechanical displacement and the other, inversion of the cavities, is an abnormal embryonal development. Parsons-Smith [102] shows that dextrocardia with complete transposition of the organs—situs inversus totalis—does not incapacitate, but incomplete transposition is usually accompanied by actual malformations of the heart and of the body. Schüssler's [103] case had supernumerary vertebrae; Moncalvi's [104] case with situs inversus and dextrocardia has congenital stenosis of pulmonary, and probably a patent ductus arteriosus; Ochsenius [105] reports the fifth case in the literature of *familial situs inversus totales* in non twins.

Goehring [106] reports a case of *anuerysm of the postaortic sinus of Valsalva* of congenital origin, which clinically gave signs of tricuspid insufficiency. Rupture resulted in death. There are only six such cases on record. Plowden [107] reports a case of congenital malformation of the heart in which there is no pulmonary artery, but there is a large abnormal branch arising from the arch of the aorta. Toy and Ellis [108] also report a case with absence of the pulmonary artery and other defects. McLean [109] has a case of congenital heart disease that cleared up in five years. He suggests that there might have been a defect in the ventricular septum, and that there has been a return to normal rather than compensation.

Weber,[110] studying sixty-two cases of congenital heart defect, finds that there is no characteristic stunting of growth or malnutrition. Children with congenital cyanosis and with dyspnea are, however, more subject to deranged nutrition.

INCIDENCE OF HEART DISEASE AND THE CARE OF CARDIAC PATIENTS

Hoffman [111] collected the statistics on heart disease. The relative frequency of valvular heart disease and endocarditis in men of military age is 2.6 per cent.; and of all forms of heart disease leading to total rejections is 3 per cent.—about the same as pulmonary tuberculosis. Conner [112] gives heart defects as causing a rejection rate of 4.25 per cent. in the army, while in 1917 the death rate in the United States registration area from heart disease was 12.1 per cent. of the total.

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102. Parsons-Smith, B.: Lancet **2**:1076 (Dec. 13) 1919.
103. Schüssler, O.: Deutsch. med. Wchnschr. **46**:658 (June 10) 1920.
104. Moncalvi, M.: Pediatria, Naples **28**:907 (Oct. 1) 1920.
105. Ochsenius, K.: Monatschr. f. Kinderh. **19**:27 (Oct.) 1920.
106. Goehring, G.: J. M. Res. **42**:49 (July-Sept.) 1920.
107. Plowden, H. H.: South Carolina M. A. J. **15**:649 (Dec.) 1919.
108. Toy, W. B., and Ellis, A. G.: J. A. M. A. **74**:322 (Jan. 31) 1920.
109. McLean, S.: J. A. M. A. **74**:1229 (May 1) 1920.
110. Weber, O.: Monatschr. f. Kinderh. **18**:205 (June) 1920.
111. Hoffman, F. L.: J. A. M. A. **74**:1364 (May 15) 1920.
112. Conner, L. A.: J. A. M. A. **74**:1564 (June 5) 1920.

There is no doubt that the cardiac patients need special care and attention. Conner outlines the growth of cardiac clinics and institutions for the care of heart cases in New York City. In 1911 there was a single outpatient heart clinic in Bellevue hospital, while in 1920 there were twenty-seven cardiac dispensary classes, accommodating 3,000 persons, convalescent homes for heart cases, inspection and care of children with crippled hearts, and special classes for these children, and occupational work under supervision of the Employment Bureau for the Handicapped. St. Lawrence and Adams [113] report in detail the care of cardiac children, with the object of making them useful citizens. Duggan [114] reports from the Employment Bureau for the Handicapped the results of placing cardiac patients and the types of employment suitable for cardiac patients. One difficulty seems to be assuring the employer that the workman with a heart lesion will not drop dead suddenly.

LESIONS OF THE AORTA

Martin [115] lists as causes for widening of the shadow of the great vessels chronic mitral lesions with dilated pulmonary artery, congenital heart lesions, long continued hypertension, a high diaphragm, arteriosclerosis and syphilitic aortitis. Brown [116] calls attention to retrosternal pain as an important symptom of acute aortic dysfunction, the prognosis of which is not necessarily grave in young persons. Stolkind [117] is unable to find in the literature a single case of proved *hereditary syphilitic aortitis* in older children, adolescents and adults, although such cases may exist. About seventy-five cases are on record of aortic changes in new-born infants with hereditary syphilis, and the pathologic findings are different from syphilitic aortitis in adults. The literature on the finding of *Spirochaeta pallida* in the tissues of children is also reviewed. Martinet [118] finds only five cases in his series of patients with aortic aneurysm whose histories exclude the possibility of syphilis.

Moon [119] reports a case of congenital stenosis of the aorta in a boy, 11 years old, who is active, energetic and bright, but somewhat undersized and pale.

THE CAPILLARIES

The conception of the capillaries has undergone a radical change in the past two years. Since about 1860, vasomotor changes have been considered synonymous with arteriomotor—a function of blood pres-

113. St. Lawrence, W. P., and Adams, E.: Hosp. Service Quart. **2**:151 (May) 1920. Quoted from Editorial, J. A. M. A. **75**:609 (Aug. 28) 1920.
114. Duggan, I. M.: Cardiac Hosp. Soc. Serv. Quart. **2**:229 (May) 1920.
115. Martin, C. E.: J. A. M. A. **74**:723 (March 13) 1920.
116. Brown, G. E.: Ann. Med. **1**:242 (July) 1920.
117. Stolkind, E. J.: Brit. J. Child. Dis. **17**:126 (Sept.) 1920.
118. Martinet, A.: Presse méd. **28**:733 (Oct. 16) 1920.
119. Moon, R. O.: Lancet **1**:1314 (June 19) 1920.

sure. The pumping mechanism [120] is not the sole factor in blood distribution—the peripheral vasomotor changes must be considered. The vasomotor control was formerly considered due to vasoconstrictor and vasodilator nerves, but more recently the possibility of chemical control—sensibility to cellular products has been discussed. Hooker [121] examines the capillaries and venules in the cat's ear, and finds that there is a definite *"capillariomotor" mechanism* which is subject to chemical and to nervous control. Baylis [122] says that the absence of a muscular coat does not rule out the possibility of active changes in the capillaries, and Krogh [123] says they function in active dilatation and there is capillary tone. In another article, Krogh [124] shows that the capillariomotor mechanism in the frog is only slightly dependent on the nerve supply, and that maintenance of the capillary tonus is dependent on the blood supply. With a lowered blood supply, the tonus diminishes, causing relaxation and the admission of a current of blood and tone is regained. The substance responsible for tonus in the blood is not known, but it is not oxygen supply. These experiments recall the old findings of Cushing on examining the capillaries in the meninges of the monkey through a trephine opening, and discovering that normally the capillaries are not continuously filled with blood, but that the filling comes in waves, or pulsations. Dale [125] accepts the view of actively contractile capillaries, and thinks this will play a part of increasing importance in the conception of the mechanism for regulating blood supply and in the explanation of shock. Wolfer [126] notes that the intrapulmonary circulation and the body circulation balance, supplement and compensate each other, and different drugs act differently on the two, epinephrin causing the vessels of the pulmonary circulation to dilate.

Danzer and Hooker [127] determine the *capillary blood pressure* with the microcapillary tonometer which fits on a microscope stage, and the pressure is applied through a capsule with a transparent membrane through which the skin is observed under the microscope with a magnification of 70x. The criterion for reading is the point where stagnation occurs. The normal pressure is 22.2 mm. Hg and there is slight diurnal variation. Kylin [128] uses the same type of apparatus and finds that in 100 healthy subjects the capillary blood pressure range is 110-190 mm. water. The arterial hypertension cases fall into two groups, one

120. Editorial, J. A. M. A. **75**:1784 (Dec. 25) 1920.
121. Hooker, D. R.: Am. J. Physiol. **54**:30 (Nov.) 1920.
122. Editorial, J. A. M. A. **74**:178 (Jan. 17) 1920.
123. Krogh, A.: J. Physiol. **52**:457, (May) 1919.
124. Krogh, A.: J. Physiol. **53**:399 (May) 1920.
125. Dale, H. H.: Bull. Johns Hopkins Hosp. **31**:256 (Aug.) 1920.
126. Wolfer, P.: Cor.-Bl. f. schweiz. Aerzte **49**:1817 (Nov. 27) 1919.
127. Danzer, C. S., and Hooker, D. R.: Am. J. Physiol. **52**:136 (May) 1920.
128. Kylin, E.: Zentralbl. f. inn. Med. **41**:505 (July 18) 1920.

with increased capillary blood pressure, and the other without any increase in the capillary pressure. The last group includes the benign nephroses.

BLOOD PRESSURE

An editorial in the *Journal of the American Medical Association* [129] calls attention to the change in the conception of the normal blood pressure: eight years ago from 150 to 160 mm. of mercury was taken as the upper limit of normal systolic blood pressure; two years later the age factor was more clearly recognized and systolic pressures over 135 mm. were considered as hypertension in persons under middle age. Alvarez [130] fixes the normal from studies made on freshmen at the University of California as from 85 to 155 mm. for women and from 90 to 175 mm. for men. In women there is a decrease in blood pressure between the ages of 17 and 25, and in men between the ages of 17 and 21. Kahn [131] shows that the normal varies with the position of the arm, both systolic and diastolic pressures decreasing as the arm is raised. For comparable results all readings should be taken with the arm at the side.

Miller [132] studies blood pressure changes of interest to surgeons.

TABLE 1.—Systolic Blood Pressure Variations Due to Position (1,000 Cases)

Position	Variation Less Than 10 Mm., per Cent.	Blood Pressure Diminished, per Cent.	Blood Pressure Increased, per Cent.	Tendency to
Dorsal	55	22.5	22.5	Remain normal
Lithotomy	64.5	11.1	24.4	Remain normal
Trendelenburg	38.7	57.2	4.1	Diminish
Combined lithotomy and Trendelenburg	25	25	50	Increase

A drop in temperature in the operating room is followed by a fall in blood pressure which is more marked when there is visceral exposure. The temperature of the operating room should never be below 70 F. in major operations. A quiet anesthesia shows no blood pressure change; a deep anesthesia is apt to give a rapid decline in blood pressure, and spinal anesthesia may give a drop that reaches the danger zone.

Cordier [133] believes that localized hypertension in the hands and feet, which occurs spasmodically without any trophic disturbance, is the first

129. Editorial, J. A. M. A. **75**:1650 (Dec. 11) 1920.
130. Alvarez, W. C.: Arch. Int. Med. **26**:381 (Oct.) 1920.
131. Kahn, M. H.: Am. J. M. Sc. **158**:823 (Dec.) 1919.
132. Miller, A. H.: J. A. M. A. **74**:514 (Feb. 21) 1920.
133. Cordier, V.: Arch. d. mal. du cœur **13**:241 (June) 1920.

stage of a phenomenon to be noted in the next ten years when this present generation will present heart and kidney conditions due to the intoxications, fatigues and latent infections of the recent war campaigns.

There is a group of three papers on *blood pressure changes in pulmonary conditions.* Amblard [134] in acute pulmonary edema finds a high blood pressure, running higher during the development; then, as the ventricle gives way, there is a sudden drop, which is followed by edema. The heart action keeps up and no arrhythmia develops. Edgeworth [135] finds that in 3 per cent. of the cases of lobar pneumonia death results from a persistent fall in blood pressure. De Bloeme,[136] studying 500 cases of pulmonary tuberculosis, finds that the blood pressure is normal in 87.7 per cent. during the first stage, in 86 per cent. during the second stage and in 75 per cent. during the third stage. Only the third stage shows a marked tendency toward a falling pressure almost 18 per cent. having subnormal pressures. Cases with a low blood pressure—from 80 to 100 mm.—all terminate fatally in less than one year.

The correlation of *endocrine dyscrasias and blood pressure changes* are studied by Engelbach [137] who analyzed 500 uncomplicated endocrine cases. Ten per cent. of the cases showed increased blood pressure, the highest pressures occurring in the 30 per cent. of these cases representing pluriglandular dycrasias; pituitary, thyroid, and ovarian changes come next in frequency as causing increased blood pressures. The prognosis in endocrine hypertension is more favorable than in vasculorenal hypertension. Downs [138] finds that splenic and thymic substances do not affect the blood pressure in dogs; ovarian and thyroid substances cause oscillation in the pressure; mammary gland substance first increases, then lowers the pressure; pancreatic substance and secretin lower the blood pressure while epinephrin raises it. Sandiford,[139] giving 0.5 c.c. of a 1:1000 solution of epinephrin chlorid gets an increase in systolic and a decrease in diastolic blood pressures. Schiff and Epstein [140] find that normal children respond to epinephrin with a considerable increase in blood pressure that is uninfluenced by change in position. Children with weak pulse and low tension may give little, if any, response to epinephrin, but if the vasomotor system is stable, the pressure rises on reclining. Children with unstable innervation usually give a prompt but slight rise in blood pressure which is not altered by

134. Amblard, L.: Paris méd. **10**:425 (May 22) 1920.
135. Edgeworth, F. H.: Bristol M.-Chir. J. **37**:86 (June) 1920.
136. de Bloeme, P. J.: Nederlandsch Tijdschr. v. Geneesk. **1**:943 (March 29) 1920; Abstr. J. A. M. A. **75**:1238 (Oct. 30) 1920.
137. Engelbach, W.: J. A. M. A. **74**:1619 (June 12) 1920.
138. Downs, A. W.: Am. J. Physiol. **52**: (July) 1920.
139. Sandiford, I.: Am. J. Physiol. **51**:407 (April) 1920.
140. Schiff, E., and Epstein, B.: Jahrb. f. Kinderh. **91**:128 (Feb.) 1920.

change in position. Lowry and Wright [141] find that the injection of epinephrin in dementia praecox cases does not give data of value in the differential diagnosis. Weber [142] calls attention to high blood pressure as a diagnostic sign in suprarenal tumors; one child 5 years old having a systolic pressure of 108 mm. Hg.

PULSE AND BLOOD PRESSURE

Sahli [143] considers that blood pressure determinations are misleading unless the pulse volume is determined at the same time. Sewall [144] suggests that estimations should be made, under different conditions, of the velocity of the blood current as represented by the product of the pulse pressure multiplied by the pulse rate.

Dawson [145] finds that physical training slows the pulse rate, increasing the diurnal variation in pulse rate; increasing the systolic pressure and decreasing the diastolic pressure thus increasing the pulse pressure. Building from Dreyer's foundation that the heart rate of the wild hare which leads an active life is 68 compared to a heart rate of 200 in the rabbit which lives an inactive life, and that wild hare's blood volume is three times the blood volume of the rabbit; and from Boney's work showing that with fatigue there is a difference in the pulse rate on standing and lying down, Schneider [146] tabulates the changes into a point system with plus and minus ratings. A score of 9 or less is characteristic of physically unfit men.

Wilson [147] tested twenty average normal children for their *circulatory reactions to graded exercises.* Immediately after exercise the pulse rate is markedly increased but in 86 per cent. it returns to normal or below normal in two minutes. The systolic pressure with moderate exercise rises to the summit in from twenty to forty seconds and falls to the pre-exercise level in two minutes. With severe exercise, the height of the rise is increased reaching a maximum in from fifty to seventy seconds, and falling more slowly, taking from three to five minutes to reach the pre-exercise level showing that the exercise tolerance of the child has been reached or passed. Mabon [148] studying changes in the blood pressure and pulse in fifty patients with effort syndrome finds that the amount of work they are able to accomplish before fatigue occurs is much less than the normal, but the pulse changes and blood pressure changes are not abnormal.

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141. Lowry, L. G., and Wright, W. W.: Boston M. & S. J. **183**:209 (Aug. 12) 1920.
142. Weber, F. P.: Practitioner, London **105**:181 (Sept.) 1920.
143. Sahli, H.: Schweiz. med. Wchnschr. **50**:2 (Jan.) 1920.
144. Sewall, H.: Am. J. M. Sc. **108**:786 (Dec.) 1919.
145. Dawson, P. M.: Am. J. Physiol. **50**:443 (Dec.) 1919.
146. Schneider, E. C.: J. A. M. A. **74**:1506 (May 29) 1920.
147. Wilson, M. G.: Am. J. Dis. Child. **20**:188 (Sept.) 1920.
148. Mabon, T. M.: Am. J. M. Sc. **158**:818 (Dec.) 1919.

TABLE 2.—Points for Grading Cardiovascular Changes
(Schneider's Table)

A. Reclining Pulse Rate		B. Pulse Rate Increase on Standing				
		0–10 Beats,	11–18 Beats,	19–26 Beats,	27–34 Beats,	35–42 Beats,
Rate	Points	Points	Points	Points	Points	Points
50– 60	3	3	3	2	1	0
61– 70	3	3	2	1	0	—1
71– 80	2	3	2	0	—1	—2
81– 90	1	2	1	—1	—2	—3
91–100	0	1	0	—2	—3	—3
101–110	—1	0	—1	—3	—3	—3

C. Standing Pulse Rate		D. Pulse Rate Increase Immediately after Exercise				
		0–10 Beats,	11–20 Beats,	21–30 Beats,	31–40 Beats,	41–50 Beats,
Rate	Points	Points	Points	Points	Points	Points
60– 70	3	3	3	2	1	0
71– 80	3	3	2	1	0	0
81– 90	2	3	2	1	0	—1
91–100	1	2	1	0	—1	—2
101–110	1	1	0	—1	—2	—3
111–120	0	1	—1	—2	—3	—3
121–130	0	0	—2	—3	—3	—3
131–140	—1	0	—3	—3	—3	—3

E. Return of Pulse Rate to Standing Normal after Exercise		F. Systolic Pressure, Standing, Compared with Reclining	
Seconds	Points	Change in Mm.	Points
0– 60	3	Rise of 8 or more	3
61– 90	2	Rise of 2–7	2
91–120	1	No rise	1
After 120: 2– 10 beats above normal	0	Fall of 2–5	0
After 120: 11– 30 beats above normal	—1	Fall of 6 or more	—1

May [149] and Lereboullet and Heitz [150] discuss the Pachon oscillometer, which gives valuable information by the amplitude of the oscillation. Lereboullet and Heitz say that it reveals apparent death in the new-born, shows maximal and minimal blood pressure, the condition of the peripheral circulation and the relative vasoconstriction of different arteries. May says the ocillometer index (amplitude of vibration) is the result of different factors, and that dividing this index by the difference between the maximal and minimal pressure gives an oscillometer ratio which is a true record of the vascular elements of the pulse.

BLOCD VOLUME

Löwy,[151] using Bangs micromethod, determines the *sodium chlorid content of capillary blood* and then injects 400 c.c. of isotonic glucose solution intravenously. Immediately afterward, and sometimes five or ten minutes later, the sodium chlorid value is again determined. Then the total blood volume $= \frac{a_2 b}{a_1 - a_2}$ where a_1 equals the salt content before infusion; a_2 the salt content after infusion; and b the amount of

149. May, E.: Presse méd. **28**:423 (June 26) 1920.
150. Lereboullet, P., and Heitz, J.: Paris méd. **10**:1 (July 3) 1920.
151. Löwy, J.: Zentralbl. f. inn. Med. **41**:337 (May 8) 1920.

fluid infused. The total blood volume normally is from one-eighteenth to one-twelfth of the total weight. Löwy gives the error of the method as not being over 5 per cent. Boenheim and Fischer [152] consider Löwy's principle as good but say that the method used for the sodium chlorid determination is not accurate enough to give even approximate results. In a series of four studies on blood volume, Hooper, Smith, Belt and Whipple; [153] Dawson, Evans and Whipple; [154] and Smith,[155] determined the soundness of the dye blood volume method which has an error of not more than 5 per cent.; and the behavior of a series of dyes, selecting vital red as the one for use. McQuarrie and Davis [156] outline a method of reading refractometrically the serum protein increase after injecting a known amount of gelatin or acacia solutions. This method has the advantage of not being affected by hemolysis, cholemia or lipemia. Harris [157] suggests substituting congo red for vital red because the sodium salt of congo red is red, and the free acid form is blue. Changing it to blue for color comparison rules out any effect from hemolysis. The optimal interval for the withdrawal of blood after injection of dye is about twice the circulation time or two and one half minutes for man. His formulas are:

$$\text{Volume of plasma} = \frac{\text{Dilution factor} \times \text{No. of C.c. of dye injected}}{\text{Ratio of strength of dye in plasma}}$$

$$\text{Volume of blood} = \text{volume of plasma} \frac{\text{100 per cent.}}{\text{Per cent. of plasma by hematocrit}}$$

$$\text{Weight of blood} = \text{volume} \times \text{specific gravity.}$$

Salvesen [158] finds that the average blood volume in six healthy young subjects is 5.95 c.c. per 1,000 gm. of body weight or from one-fourteenth to one-nineteenth of the body weight.

Plesch [159] determines the *volume of blood discharged during phases of the heart cycle* by the amount of oxygen given off by arterial blood. The oxygen consumption is determined by the respiratory metabolism; the amount of oxygen held in the blood by a special type of hemoglobinometer, and then the oxygen content of blood from heart by the rubber bag apparatus.

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152. Boenheim and Fischer: Zentralbl. f. inn. Med. **41**:553 (Aug. 7) 1920.
153. Hooper, C. W.; Smith, H. P.; Belt, A. E., and Whipple, G. H.: Am. J. Physiol. **51**:205 (March) 1920.
154. Dawson, A. B.; Evans, H. M., and Whipple, G. H.: Am. J. Physiol. **51**:232 (March) 1920.
155. Smith, H. P.: Ibid. p. 221.
156. McQuarrie, I., and Davis, N. C.: Ibid. p. 257.
157. Harris, D. T.: Brit. J. Exper. Path. **1**:142 (June) 1920.
158. Salvesen: J. Biol. Chem. **40**:109 (Nov.) 1919.
159. Plesch, J.: Deutsch. med. Wchnschr. **45**:1401 (Dec. 18) 1919.

BLOOD CONCENTRATION

Rubow [160] by restricting the fluid intake to about one-third of the twenty-four hour requirement, reduces the water content of the blood as much as 12.5 per cent. in a few days. Rominger [161] finds that the water content in the blood of infants is remarkably stable. Water passes readily from the stomach to the blood, but with the exception of decomposition cases the hydremia is transient.

Blood concentration means a failing circulation, an inefficient oxygen distribution, oxygen starvation, decreased temperature and finally death. In influenza, while edema of the lungs is indirectly concerned, Underhill and Ringer [162] believe that blood concentration is the immediate cause of death. The hemoglobin content is used as an index of concentration, and treatment to combat the concentration should be begun before the blood concentration has reached 125 per cent. of the individuals normal hemoglobin value. Wilson and Goldschmidt [163] believe that the loss of water from the blood in the development of lung edema is the cause of blood concentration and not imbibition of water by tissues as a result of oxygen want. Hill's [164] views oppose those of Wilson and Goldschmidt, but stagnation is considered the factor in causing oxygen want.

Lutz and Schneider [165] find that the cardiac and respiratory centers are stimulated by a decrease in oxygen and inhibited by an increase in oxygen. The heart responds in from five to fifty-five seconds to an oxygen decrease. Barcroft [166] divides *anoxemia* into three types, (a) the anoxic type with general condition low and low oxygen pressure, (b) the anemia type gaged by the quantity of oxygen liberated from the blood, and (c) the stagnant type with increased hemoglobin.

THE ACID-BASE EQUILIBRIUM AND ALKALI RESERVE

Studies on the alkali reserve and the acid-base equilibrium have been fewer in number during the past year and there has been more of an attempt made to establish normal and comparable values. Collip [167] finds that alkali reserve is either unaltered or diminished during sleep. Haskins and Osgood [168] use the Van Slyke titration method as a class room method, but find that students have difficulty in matching a clear

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160. Rubow, V.: Hospitalstidende **63**:305 (May 19) 1920; Abstr. J. A. M. A. **75**:514 (Aug. 14) 1920.
161. Rominger, E.: Ztschr. f. Kinderh. **26**:23 (July 28) 1920.
162. Underhill, F. P., and Ringer, M.: J. A. M. A. **75**:1531 (Dec. 4) 1920.
163. Wilson, D. W., and Goldschmidt, S.: Am. J. Physiol. **50**:157 (Oct.) 1919.
164. Hill, L.: Lancet **1**:359 (Feb. 14) 1920.
165. Lutz, B. R., and Schneider, E. C.: Am. J. Physiol. **50**:327 (Dec.) 1919.
166. Barcroft, J.: Lancet **2**:485 (Sept. 4) 1920.
167. Collip, J. B.: J. Biol. Chem. **41**:473 (April) 1920.
168. Haskins, H. D., and Osgood, E. F.: J. Lab. & Clin. M. **6**:37 (Oct.) 1920.

phosphate standard with the turbid plasma, so they add raw starch to the standard, making it also turbid, and the students get better results. Haggard,[169] in a paper forming a portion of the studies on hemato-respiratory functions, reports that an increase in the body temperature lowers the alveolar carbon dioxid tension and reduces the amount of carbon dioxid in the blood. No compensatory changes occur in the carbon dioxid combining power of the blood. The alkali in use is not lowered in proportion to the decrease in the carbon dioxid dissolved so that the C_2H_2O: $NaHCO_3$ ratio is lowered and presumably the C_H value is lowered. Haggard and Henderson,[170] in three papers in the same series of studies, find that with low oxygen, over-breathing occurs before the blood alkali is appreciably reduced. Intravenous injection of lactic acid does not induce an acidosis at all commensurate with the amount administered, and an increase in the lactates in the blood or urine is probably an indication of a low C_2H_2O: $NaHCO_3$ ratio. Over-breathing throws off an excess carbon dioxid bringing the carbon dioxid ratio below normal. This alkalosis is compensated by the disappearance of alkali from the blood. The process is reversed by normal oxygen tension and acidosis recalls the alkali to the blood. The barometric pressure to which one is acclimated is the fundamental factor controlling the volume of air breathed per unit mass of carbon dioxid eliminated, the alveolar carbon dioxid tension, and the amount of alkali called into use in the blood. Haggard [171] finds that the corrections in the Henderson-Morris method for determining carbon dioxid in plasma and whole blood almost exactly neutralize each other and the gas burette readings may be taken direct each 0.01 c.c. = 1 volume per cent. of carbon dioxid.

Collip and Backus [172-173] finds that the carbon dioxid combining power of the plasma is an approximate index of the alkali reserve of the body fluids in normal animals. The C_H of the spinal fluid is protected to a greater degree than that of blood; the carbon dioxid combining power of spinal fluid as compared with plasma is at a high level in shock. Forced breathing washes out the carbon dioxid from the blood, but the tissues give a buffer reaction, taking up alkali, which accounts for the rapid return to normal when the forced breathing stops. The tetany and muscle cramps developing during hyperpnea would appear to be due to tissue alkalosis. Grant and Goldman's [174] experiments seem to substantiate this view.

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169. Haggard, H. W.: J. Biol. Chem. **44**:131 (Oct.) 1920.
170. Haggard, H. W., and Henderson, Y.: J. Biol. Chem. **43**:15 (Aug.) 1920.
171. Haggard, H. W.: J. Biol. Chem. **42**:237 (June) 1920.
172. Collip, J. P., and Backus, P. L.: Am. J. Physiol. **51**:551 (April) 1920.
173. Collip, J. P., and Backus, P. K.: Ibid. p. 568.
174. Grant, S. B., and Goldman, A.: Am. J. Physiol. **52**:209 (June) 1920.

The *effect of hemorrhage on the alkali reserve* is studied by Buell [175] who finds that the drop in alkali reserve is slight if the animal is quiet; and by Tatum [176] who believes that a change in the acid-base balance in the body cells is responsible for rise in blood sugar after hemorrhage. *Ether anesthesia affects alkali reserve.* Carter [177] finds that the carbon dioxid combining capacity of dog's blood diminishes after the first hour of anesthesia, the amount being in direct proportion to the duration of the anesthetic, and the diminished alkali reserve continues for from one-half to one hour after the anesthetic. Prentice, Lund and Harbo [178] give the figures for alkali reserve as 0.23 before ether falling from 0.016 to 0.018 with ether. Raymond [179] finds that the fall in alkali reserve is relatively insignificant in acute shock, but with late shock the plasma may show a high alkali reserve. The condition of the animal cannot be judged by the alkali reserve. Adams and Sturges [180] find in fifty-four cases of effort syndrome that the carbon dioxid falls within normal limits, the combining power of the blood is normal and there is no indication that there is a decrease in the buffer salts of the blood.

The acid-base equilibrium of the blood is disturbed in acute tubular nephropathies. MacNider [181] thinks this shows the tubular part to be more important in the maintenance of acid base equilibrium than the vascular portion. With experimental infections Hirsch [182] finds that the blood alkali reserve is lowered at the time of the initial leukocytosis, then rises to, or above, the original level. In epidemic influenza and bronchopneumonia, Hachen and Isaacs [183] consider a comparison of the alkali reserve and the temperature as important in prognosis. With an alkali reserve of 46 or lower, the prognosis is good. With an alkali reserve of from 46 to 52, the prognosis is indefinite. With an alkali reserve of 53 and over, the prognosis is good. Sullivan and Stanton,[184] studying the alkali reserve in pellagra find little uncompensated acidosis. Henderson and Haggard [185] accelerate the elimination of carbon monoxid in carbon monoxid asphyxia by using a mixture of carbon dioxid and oxygen.

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175. Buell, M. V.: J. Biol. Chem. **40**:29 (Nov.) 1919.
176. Tatum, A. L.: J. Biol. Chem. **41**:59 (Jan.) 1920.
177. Carter, W. S.: Arch. Int. Med. **26**:319 (Sept. 15) 1920.
178. Prentice, W.; Lund, H. O., and Harbo, H. G.: J. Biol. Chem. **44**:211 (Nov.) 1920.
179. Raymond, B.: Am. J. Physiol. **53**:109 (Aug.) 1920.
180. Adams, F. D.; Sturgis, D. C., and Sturges, C. C.: Am. J. M. Sc. **158**: 816 (Dec.) 1919.
181. MacNider, W. de B.: Arch. Int. Med. **26**:1 (July) 1920.
182. Hirsch, E. F.: J. A. M. A. **75**:1204 (Oct. 30) 1920.
183. Hachen, D. S., and Isaacs, R.: J. A. M. A. **75**:1624 (Dec. 11) 1920.
184. Sullivan, M. X., and Stanton, R. E.: Arch. Int. Med. **26**:41 (July) 1920.
185. Henderson, Y., and Haggard, H. W.: J. Pharmacol. & Exper. Therap. **16**:11 (Aug.) 1920.

FERMENTS IN BLOOD

Lewis and Mason [186] find no constant relation between the variation in level of the blood ferments in nephritis and the progress of the disease. Fujimoto [187] thinks that diastatic substance passes from the pancreas to the liver where it is mixed with blood and lymph and its output is regulated by the liver. This is based on the fact that hepatotoxin decreases the diastase content of peripheral blood, while pancreatoxin and neurotoxin have no effect. Catalases, according to Reimann and Becker,[188] decrease in the blood in 65 per cent. of the cases during anesthesia. In anemia, Krumbhaar and Musser [189] find the catalase index (the catalase content divided by the hemoglobin content or the red blood cell count) of value in spite of wide individual variation. In anemias the catalase index varies from 15 to 53, with an average of 37+.

NITROGEN IN THE BLOOD

The study of nitrogen distribution in the blood of children results in the establishment of a better understanding of the normal. Utheim [190] finds that the serum protein in normal infants is from 6 to 6.5 per cent. until the tenth or eleventh month, when the protein begins to rise and reaches the adult level of about 8 per cent. by the fifteenth month. In athreptic and premature infants the protein may be as low as 4 per cent. Chapin and Meyers [191] report the findings on chemical examination of the blood of 149 children, including thirty-eight nephritics and six diabetics. They are similar to adults but the kidney of a child is more efficient, and nephritis does not result so quickly in urea retention as in adults. Stransky [192] examined the residual nitrogen in eighty-five children, including fifty infants. The high nitrogen content of the blood of the newborn has no clinical significance and diminishes by the end of the first week, after which the findings scarcely differ from those of early childhood. The values range from 40 to 50 mg. per 100 gm. of blood. Hammett [193] says that while total nitrogen, nonprotein nitrogen and sugar in the blood vary in the same person from week to week, the sum of the average deviation of the constituents for any given individual may be the *index of metabolic stability* for that person.

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186. Lewis, D. S., and Mason, E. H.: J. Biol. Chem. **44**:455 (Nov.) 1920.
187. Fujimoto, B.: Am. J. Physiol. **50**:208 (Nov.) 1919.
188. Reimann, S. P., and Becker, C. E.: Am. J. Physiol. **50**:56 (Oct.) 1919.
189. Krumbhaar, E. B., and Musser, J. H.: J. A. M. A. **75**:104 (July 10) 1920.
190. Utheim, K.: Am. J. Dis. Child. **20**:366 (Nov.) 1920.
191. Chapin, H. D., and Meyers, V. C.: Am. J. Dis. Child. **18**:555 (Dec.) 1919.
192. Stransky: Monatschr. f. Kinderh. **19**:10 (Oct.) 1920.
193. Hammett, F. S.: J. Biol. Chem. **41**:599 (April) 1920.

Smith, Belt and Whipple [194] call attention to the remarkable stability of *serum protein concentration.*[195] When serum protein is depleted by interval bleeding and reinjection of washed corpuscles, there is an emergency reaction in fifteen minutes, then a gradual rise during the first twenty-four hours, and a more sluggish increase the next few days. The physiologic value of serum protein is not understood, but it may be a stabilizing value. With too great a dilution of serum proteins there is cell injury and clinical shock.[196]

Cullen and Van Slyke [197] partition the fibrin, globulin and albumin in the blood by means of nitrogen determinations. The fibrin nitrogen is determined directly, and globulin and albumin nitrogen are determined from the figures for total nitrogen, nonprotein nitrogen and fibrin nitrogen. In *glomerulonephritis and arteriosclerotic nephritis* Fitz [198] finds no relationship between the nonprotein nitrogen in the blood, the phenolsulphonephthalein test and the type of nephritis. Jacobson and Edwards [199] say the blood urea level is governed by previous diet, fluid retention, and unknown metabolic factors and is not a reliable index of nitrogen retention.

In anesthesia and after operations Reiman and Hartman [200] find blood urea and nonprotein nitrogen increased in all cases. Martin and Denis,[201] studying the chemical changes in the blood following roentgen-ray treatment of *leukemia* report that there is no connection between the white count and the uric acid concentration. In cases with a high nonprotein nitrogen there is a gradual fall, creatinin values are normal, urea is low, and uric acid is persistently high. Martin and Denis suggest that in leukemia there is a nitrogen compound in the white cells not accounted for in the present methods of microblood analysis.

In *pernicious anemia* and *severe anemias* Peters and Rubnitz [202] report that the nitrogen index $\frac{\text{per cent. whole blood nitrogen}}{\text{per cent plasma nitrogen}}$ is lowered and it may be used to indicate the state and progress of the anemic condition. Gettler and Lindeman [203] give figures for nonprotein nitrogen, urea nitrogen, amino-acid nitrogen, uric acid and creatinin in the blood obtained from thirty-two cases of *pernicious anemia.* Aminoacid

194. Smith, H. P.; Belt, E. A., and Whipple, G. H.: Am. J. Physiol. **57**: 54 (May) 1920.
195. Smith, H. P.; Belt, E. A., and Whipple, G. H.: Ibid. p. 72.
196. Smith, H. P.; Belt, E. A., and Whipple, G. H.: Ibid. p. 101.
197. Cullen, G. E., and Van Slyke, D. D.: J. Biol. Chem. **41**:587 (April) 1920.
198. Fitz, R.: Boston M. & S. J. **183**:247 (Aug. 26) 1920.
199. Jacobson, A. T. B., and Edwards, H.: Am. J. M. Sc. **159**:833 (June) 1920.
200. Reimann, S. P., and Hartman, F. L.: Am. J. Physiol. **50**:56 (Oct.) 1919.
201. Martin, C. L., and Denis, W.: Am. J. M. Sc. **160**:223 (Aug.) 1920.
202. Peters, A. W., and Rubnitz, A. S.: Arch. Int. Med. **26**:561 (Nov.) 1920.
203. Gettler, A. O., and Lindeman, E.: Arch. Int. Med. **26**:453 (Oct.) 1920.

nitrogen is always increased, and ninety per cent. of cases have increased uric acid, while the serum proteins may be reduced from 40 to 50 per cent.

In *influenzal pneumonia,* Wells [204] finds most fatal cases show an increase of blood uric acid, the average being 4.45 mg. per 100 c.c. of blood as contrasted with an average in the nonfatal cases of 3.14 mg. per 100 c.c. Pemberton and Foster [205] find a high blood creatinin in 50 per cent. of the cases of *arthritis* which in some cases declines with improvement. Dufour and Semelaigne,[206] in a case of *epileptiform seizures* after injections of neo-arsphenamin find an increase in the blood urea from 0.25 to 0.40 at normal times to 0.84 a few hours before a seizure. Loeper, Thinj and Tonnet [207] report profound alterations in the nitrogen equilibrium of the blood with residual nitrogen increased and urea nitrogen decreased in cancer patients. The frequent increase in blood urea without renal change is significant.

BLOOD SUGAR

Methods for the accurate estimation of blood sugar are still unsettled. The work of Cowie and Parsons [208] shows that the picrate solution used in the Lewis-Benedict method is sensitive to epinephrin in 0.025 mg. per cent., to acetone in blood solutions but not in water solutions, to diacetic acid and to creatinin. Creatinin does not often interfere. They suggest that the hyperglycemia occurring in emotional states may be due to epinephrin thrown into the circulation without mobilizing the glycogen stores in the liver. Rassero [209] finds both crystalloid and colloid elements in the serum from cattle, sheep, rabbits and guinea-pigs that produce vasoconstricting action on intestinal muscle, uterine muscle of cats and artery muscles in cattle. These substances resemble epinephrin. Lippmann [210] finds that the drop in the freezing point in diabetes does not parallel the intensity of the glycemia, and this indicates disturbance in the mechanism regulating the osmotic tension of the blood, and that other reducing substances beside glucose are present.

Feigl [211] modifies Bangs' micromethod for blood examination obtaining results with extracts from as small a quantity as 200 mg. of blood

204. Wells, C. W.: Arch. Int. Med. **26**:443 (Oct.) 1920.
205. Pemberton, R., and Foster, G. L.: Arch. Int. Med. **25**:243 (March) 1920.
206. Dufour, H. ,and Semelaigne, G.: Bull. et mém. Soc. méd. d. hôp. de Par. **44**:58 (Jan. 16) 1920.
207. Loeper, Thinj, and Tonnet: Progrès méd. **35**:159 (April 10) 1920.
208. Cowie, D. M., and Parsons, J. P.: Arch. Int. Med. **26**:333 (Sept.) 1920.
209. Rassero, J. R. F.: Nederlandsch Tijdschr. v. Geneesk. **1**:785 (March 6) 1920.
210. Lippmann, A.: Zentralbl. f. inn. Med. **41**:41 (Jan. 17) 1920.
211. Feigl, J.: Zentralbl. f. inn. Med. **41**:17 (Jan. 10) 1920.

and using picrate reduction for sugar. Silvestri and Aiello [212] object to Bangs' micromethod and offer a modification of Bertrand's method after adding a known amount of glucose to the solution. Wallis and Gallagher [213] give a new microchemical method for estimation of sugar in blood.

Mertz [214] analyzes the literature and decides that the carbohydrate metabolism of infants and young children obeys the same laws as adults. Strause [215] finds variations in the normal blood sugar due to weather conditions, all the curves in a group of individuals being higher in March than in September. Water intake and excretion do not affect blood sugar. Alimentary hyperglycemia reaches its height in normals in half an hour and descends at once, while in diabetes the height is reached in one hour, and descends slowly. Brösamlen and Sterkel [216] find a marked reduction in blood sugar after physical exertion. Asphyxial blood transfused into rabbits, in Yamakami's [217] experiments causes a rise in the blood sugar content, which is not due to the sugar in the asphyxial blood injected. Delatour [218] thinks that the pancreas produces substances that favor metabolism of sugar which explains the hyperglycemia in depancreatized dogs. Ross and Davis [219] conclude that the hyperglycemia with ether is due to reduction of the influence of the internal secretion of the pancreas. Rhodenburg and Pohlman [220] think that the hyperglycemia following injection of fats and polypeptids which are supposed not to produce antibody production, is protective and may be a test to check antibody production when neither precipitin, agglutinins or lysins can be demonstrated. Friedenwald and Grove [221] find that carcinomia of the gastrointestinal tract gives a characteristic blood sugar tolerance curve, whether cachexia exists or not.

BLOOD LIPOIDS

Burns [222] thinks that the origin of xanthoma multiplex is an increase in the blood cholesterin with deposits of cholesterin in the skin.

Schippers [223] questions whether the low fat content of the blood (about half the normal content) in infants with exudative diathesis

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212. Silvestri, S., and Aiello, G.: Policlinico, Rome **27**:643 (June 21) 1920.
213. Wallis, R. L. M., and Gallagher, C. D.: Lancet **2**:784 (Oct. 16) 1920.
214. Mertz, A.: Arch. f. Kinderh. **68**:254 (Oct. 16) 1920.
215. Strause, S.: Arch. Int. Med. **26**:751 (Dec.) 1920.
216. Brösamlen, O., and Sterkel, H.: Deutsch. Arch. f. klin. Med. **130**:358 (Oct. 24) 1919.
217. Yamakami, K.: Am. J. Physiol. **50**:177 (Nov.) 1919.
218. Delatour, B. J.: Arch. Int. Med. **25**:1542 (April) 1920.
219. Ross, E. L., and Davis, L. H.: Am. J. Physiol. **53**:391 (Oct.) 1920.
220. Rhodenburg, G. L., and Pohlman, H. E.: Am. J. M. Sc. **159**:853 (June) 1920.
221. Friedenwald, J., and Grove, G. H.: Am. J. M. Sc. **160**:313 (Sept.) 1920.
222. Burns, F. S.: Arch. Dermat. and Syph. **2**:415 (Oct.) 1920.
223. Schippers, J. C.: Nederlandsch Tijdschr. v. Geneesk. **2**:1081 (Sept. 18) 1920; Abstr. J. A. M. A. **75**:1530 (Nov. 27) 1920.

may not throw light on the nature of the anomaly. Strathmann-Herwig [224] finds that breastfed and artificially fed infants who are normal have the same cholesterin content in the blood serum. Cholesterin is usually increased in infections and decreased in anemia and infantile scurvy. Richter-Quittner [225] says that normally cholesterin is divided about equally between the blood plasma and the corpuscles. Free cholesterin occurs only in the corpuscles, and cholesterin esters in the plasma, but with disease processes, free cholesterin may occur in the plasma also together with the cholesterin esters. Chaffard, Laroche and Grigaut [226] report a marked drop in cholesterin in the blood during the febrile states, with a high rise after defervescence in moderate cases, but no rise in mild or severe cases. Cholesterin probably has an antitoxic action and presides over the antibody formation. Hypercholesterinemia occurs in chronic nephritis and may be a differential diagnostic point ruling out cardiac edema. Henes [227] finds hypercholesterinemia usually in cases of chronic nephritis that are progressing favorably. He also assigns a protective rôle to cholesterol. Blood lipoids play a part analogous to antitoxin. They seem to act as a protection and to counteract certain poisonous substances. Beumer [228] notes that the hypercholesterinemia in nephroses is independent of edema and fever and could not be influenced by restriction of fat or lipoid in the diet.

Kipp [229] finds that transfusion of whole blood does not alter the cholesterol content in the blood in cases of pernicious anemia, and in pneumonia he finds a primary hypocholesterinemia dependent in degree on the activity of the leukocytes, a secondary hypercholesterinemia and the return to normal.[230] The variations are dependent on the leukocytes, and the lipoids act as an antitoxic substance.

HYPERINDICANEMIA

Van Vloten [231] tabulates the indican and the urea content of the blood serum from forty patients showing the variations with diet and the importance of blood indican for prognosis. The normal range limits by different observers run from 1.4 to 1.8 mg. per liter. In uremia,

224. Strathmann-Herwig: Monatschr. f. Kinderh. **19**:20 (Oct.) 1920.
225. Richter-Quittner, M.: Arch. f. inn. Med. **1**:425 (June 1) 1920.
226. Chaffard, A.; Laroche, G., and Grigaut, A.: Ann. de Méd. **8**:69 (Aug.) 1920.
227. Henes, E.: Arch. Int. Med. **25**:411 (April) 1920.
228. Beumer, H.: Arch. f. Kinderh. **68**:64 (Aug. 31) 1920.
229. Kipp, H. A.: J. Biol. Chem. **43**:383 (Sept.) 1920.
230. Kipp, H. A.: J. Biol. Chem. **44**:215 (Nov.) 1920.
231. Van Vloten, W. J. Van B.: Nederlandsch Tijdschr. v. Geneesk. **2**:585 (Aug. 14) 1920; Abstr. J. A. M. A. **75**:1170 (Oct. 23) 1920.

the urea and the indican content do not run parallel, although there is usually indicanemia; when it is persistently high, even though the urea content is normal, the prognosis is grave.

INORGANIC CONSTITUENTS IN BLOOD

Scheer [232] finds that the *chlorid level in the blood* serum of infants is dependent on the secretion of gastric juice. During digestion the chlorin level declines; to rise after the stomach is emptied. Host,[233] using Bangs' micromethod, finds that the chlorids exist chiefly in the serum and less in the corpuscles. Fredericia [234] confirms Van Slyke's work on the partition of chlorids between the plasma and the corpuscles. Rodillon,[235] and Meyers and Short [236] give new modifications or methods for determining the chlorids in blood. Rodillon calls the chlorids in the blood the index of kidney condition.

Denis and Minot [237] study the *phosphate retention in the blood in nephritis.* In 65 per cent. of the nephritis cases there was definite retention of inorganic phosphates in the plasma. In eleven fatal cases there was a premortal rise to as much as ten times the normal value.

The *calcium content of the blood* is still a matter of great interest. An editorial [238] in the *Journal of the American Medical Association* summarizes the uses of calcium in bones, in the clotting of blood and milk, in the maintenance of physiologic equilibrium, in the preservation of normal vessel permeability, and in the normal irritability of parts of the nervous system. Stheeman and Arntzenius [239] consider the "calcipriva stigma" the basis for spasmophilia, habitus asthenicus and universal asthenia. In fifty-eight healthy children the calcium content of the blood was constantly between 12 and 13 mg. per 100 c.c. of serum; in six children the range was from 8.25 to 17 mg. per 100 c.c. of serum.[240] The figures are low in prerachitic conditions, intestinal infantilism, tuberculosis and asthenia, the severity of the condition being reflected in the decrease in calcum. Erb's sign is nearly always positive with a low calcium content, becoming more and more positive as the calcium deficit becomes greater. Chvostek's sign has the same significance. In rachitis the calcium content in the blood may be high, but the

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232. Scheer, K.: Jahrb. f. Kinderh. **91**:347, 1920.
233. Host, H. F.: J. Lab. & Clin. M. **5**:713 (Aug.) 1920.
234. Fredericia, L. S.: J. Biol. Chem. **42**:245 (June) 1920.
235. Rodillon: Presse méd. **28**:85 (Jan. 31) 1920.
236. Meyers, V. C., and Short, J. J.: J. Biol. Chem. **44**:47 (Oct.) 1920.
237. Denis, W., and Minot, A. S.: Arch. Int. Med. **26**:99 (July) 1920.
238. Editorial, J. A. M. A. **75**:941 (Oct. 2) 1920.
239. Stheeman, H. A., and Arntzenius, A. K. W.: Nederlandsch Tijdschr. v. Geneesk. **1**:1030 (March 27) 1920.
240. Stheeman, H. A., and Arntzenius, A. K. W.: Nederlandsch Tijdschr. v Geneesk. **1**:1168 (April 3) 1920.

calcium in the tissues is actually deficient. It is evident from the positive Erb's sign that there is more than one kind of calcium in the blood, one efficient for growth and one nonefficient. The calcium content of the serum is important, but the calcium balance does not tell whether there is calcium impoverishment or calcium abundance until combined with nerve signs. Handowsky [241] is unable to find any difference in the calcium range in adults with a positive Chvostek sign and adults without a positive Chvostek. Jacobowitz [242] finds children free from tetany have a higher calcium level in the blood than children with tetany.

Kramer and Howland [243] show that the calcium in the blood serum as determined by their method is remarkably constant in adults, the normal content being from 9 to 10 mg. per 100 c.c. of serum. The decrease in tetany may be more than 50 per cent.

Denis and Minot,[244] and Clark [245] substantiate the findings that calcium by mouth, or intravenously or subcutaneously and the feeding of diets rich in calcium does not make any appreciable difference in the calcium content of bones, blood and other tissues. Stheeman and Arntzenius [239] point out that cod liver oil and phosphorus promote the retention of calcium in the body. Krehbiel [246] finds that the blood calcium is within normal limits in cancer and obliterating arteritis, showing that calcium metabolism is not a factor in cancer.

TESTS FOR OCCULT BLOOD

Penn [247] makes a smear of feces on a microscope slide, spreads two or three drops of glacial acetic acid over the smear, and heats gently over a flame. On the addition of one or two drops of benzidin dissolved in alcohol and the same amount of hydrogen peroxid, a dark blue color develops almost immediately if blood be present. The method commends itself as rapid and simple. Boas [248] says the reagents are often too strong in the benzidin test. He used 0.5 per cent. benzidin solution and in place of hydrogen peroxid he used barium dioxid which is more stable. With the test used in this way very slight hemorrhages may be missed.

RED BLOOD CORPUSCLES

The *hemoglobin content* shows a diurnal variation which commonly reaches 10 per cent. but may reach 30 per cent. (Dreyer, Bazett and

241. Handowsky, I.: Jahrb. f. Kinderh. **91**:432, 1920.
242. Jocobowitz, S.: Jahrb. f. Kinderh. **92**:256, 1920.
243. Kramer, B., and Howland, J.: J. Biol. Chem. **43**:35 (Aug.) 1920.
244. Denis, W., and Minot, A. S.: J. Biol. Chem. **41**:357 (March) 1920.
245. Clark, G. W.: J. Biol. Chem. **43**:89 (Aug.) 1920.
246. Krehbiel, O.: J. Cancer Res. **5**:199 (April) 1920.
247. Penn, S.: Med. J. Australia **1**:525 (June 5) 1920.
248. Boas, I.: Berl. klin. Wchnschr. **56**:939 (Oct. 6) 1919.

Pierce,[249]) the excursions being more marked with a low hemoglobin content. McEllroy [250] uses the fact that potassium ferricyanid does not liberate oxygen from methemoglobin but does from hemoglobin to determine the amount of methemoglobin. Total hemoglobin is determined, then the oxygen capacity and the difference gives the methemoglobin. Hemoglobin in itself has been held to be toxic, but Bayliss [251] finds that hemolysis in itself does not cause injury, hemoglobin is harmless when circulating free in plasma, and can carry oxygen. The results of the transfusion of incompatable blood are probably due to the action of a foreign protein.

To study the blood of animals in vitro, Normet [252] injects sodium citrate into the circulation. He demonstrates to his own satisfaction the development of blood corpuscles, eosinophils and erythrocytes, direct from the mononuclear leukocytes by a process of budding which is a method of cell reproduction not suspected in metazoa. Rieux [253] gives the "genealogic tree of the blood corpuscle" showing additional stages preceding the mature erythrocytes and polymorphonuclears. The presence of the normomegaloblastic reaction, of nuclear relics and basophil granular erythrocytes are signs of recuperation of blood forming elements, and signs that the bone marrow is capable of producing blood corpuscles. Bockhoven [254] thinks that basophil inclusions in the erythrocytes must be nuclear remnants and are signs of grave import. Walterhöfer [255] says their pathologic significance lies not in the inclusion bodies in the erythrocytes but in the fact that red cells with azurophil inclusion bodies appear in the peripheral circulation. They are new cells, traceable to some bone marrow reaction. Cunningham [256] suggests that the reticulated net work in the red blood cells may give a clue to the age of the cell, those with reticulation being immature and being an index to the hematopoietic activity. To stain the reticulum, smear a small drop of a 0.5 per cent. solution of brilliant cresyl blue over a small area on the slide, let dry, then wipe off the thick margin. Take a fresh blood drop on a cover slip, drop face down over the area of stain, make a smear and dry. Stain with Wright's stain. The reticulum is deep or light blue. Bauer and Buschner [257] testing the "resistance

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249. Dreyer, G.; Bazett, H. C., and Pierce, H. F.: Lancet **2**:588 (Sept. 18) 1920.
250. McEllroy, W. S.: J. Biol. Chem. **42**:297 (June) 1920.
251. Bayliss, W. M.: Brit. J. Exper. Path. **1**:1 (Feb.), 1920.
252. Normet: Bull. de l'Acad. de méd. Par. **83**:163 (Feb. 24) 1920; Abstr. J. A. M. A. **74**:1489 (May 22) 1920.
253. Rieux, J.: Arch. d. mal. du cœur **13**:254 (June) 1920.
254. Bockhoven, M.: Ztschr. f. klin. Med. **89**:304, 1920.
255. Walterhöfer, G.: Deutsch. med. Wchnschr. **46**:116 (Jan. 29) 1920.
256. Cunningham, T. D.: Arch. Int. Med. **26**:405 (Oct.) 1920.
257. Bauer, J., and Buschner: Deutsch. Arch. f. klin. Med. **130**:172 (Sept. 26) 1919.

range" of erythrocytes to varying concentration of salt solution find the widest range in severe anemias, and the smallest range in mild tuberculosis. The range varies in the same person at different times, and the younger erythrocytes have the highest resistance.

Craik [258] uses doses of lead by mouth to produce polychromatic and punctated red corpuscles. These marked discs in the circulation are young corpuscles, and they are means of tracing malarial parasites. Multiple infections of the marked discs suggest that the youngest corpuscles are the least resistant to the plasmodium.

Wyss [259] presents evidence that in the blood circulation erythrocytes are round or egg shaped, but that they collapse with the escape of the oxygen when the blood is drawn. Taylor [260] finds that most of the red blood corpuscles injected subcutaneously do not reach the circulation as cells, although the hemoglobin is raised by such an injection.

Kambe and Komiya [261] conclude that preserved erythrocytes may retain their physiologic properties and live a normal length of life after transfusion. In transfusion after hemorrhage, nucleated forms may be absent so that the newly supplied cells prevent stimulation of the blood forming organs. In a blood effusion in a cadaver exhumed seven months after death, Straussmann [262] finds the red blood corpuscles unchanged, and well preserved, the lymphocytes present but altered, and the polymorphonuclears gone completely.

The sedimentation or sinking velocity of blood corpuscles in paresis is increased to about one-sixth of the time required in dementia praecox. Plant [263] says that acceleration of sedimentation is a pathologic sign whose significance has not been determined, but the acceleration in pregnancy may have a diagnostic value. Linzenmeier [264] finds that the sedimentation time for corpuscles in the blood of nonpregnant women is five or six hours, while in pregnant women from the fourth month on, the time is less than two hours, decreasing to fifty minutes in the last three months.

Goodman [265] finds an increase in red blood cells following inflation of the peritoneal cavity with oxygen; that this may be due to the mechanical effect of pressure on venous trunks is shown by the fact that the same effect can be produced in rabbits by nitrogen inflation.

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258. Craik, R.: Lancet **1**:1110 (May 22) 1920.
259. Wyss, O.: Schweiz. med. Wchnschr. **50**:226 (March 18) 1920.
260. Taylor, R.: Am. J. Dis. Child. **20**:337 (Oct.) 1920.
261. Kambe, H., and Komiya, E.: Am. J. Physiol. **53**:1 (Aug.) 1920.
262. Straussmann, G.: Berl. klin. Wchnschr. **56**:994 (Oct. 20) 1919.
263. Plant, F.: München. med. Wchnschr., March 5, 1920, p. 279.
264. Linzenmeier, G.: Zentralbl. f. Gynäk. **44**:816 (July 24) 1920.
265. Goodman, C.: J. A. M. A. **74**:1515 (May 29) 1920.

Gregg, Lutz and Schneider [266] contrast the theories of blood concentration versus a dormant supply of erythrocytes as the cause of fluctuations in the distribution of hemoglobin and erythrocytes under states of low oxygen tension. The question seems to have been settled in a satisfactory manner by records taken on the summit of Pike's Peak and at Cripple Creek during the summer of 1903 when comparisons between the curves of the red corpuscle increase, the hemoglobin and the specific gravity from daily records on six normal healthy individuals, show that the increase and daily fluctuation cannot be accounted for by different degrees in blood concentration (Kemp[267]) and must be due to a storage of mobile red corpuscles.

POLYCYTHEMIA

Thaysen [268] makes systematic red counts over a long period of time in polycythemia. There are remarkable fluctuations of as much as 5,000,000 in a period of twenty-four hours. This is explained by varying contraction and dilation of skin capillaries due to an unstable vasomotor system. An editorial [269] in the *Journal of the American Medical Association* states that the real factor in polycythemia is unknown, that splenic tuberculosis as a cause has been abandoned. There are two accepted causes, for true polycythemia, one hyperplasia of the erythroblastic bone marrow, and the other a decrease in the rate of destruction of the red corpuscles. Lamson [270] says that *acute polycythemia* is always due to blood concentration, no matter whether it be physiologic, as the polycythemia in emotional states, or produced by local irritation or the injection of histamin. Bing [271] reports four cases of polycythemia with duodenal ulcer. The polycythemia here may be due to abnormal loss of sodium chlorid.

Weber[272] classifies the *secondary forms of polycythemia rubra* as: (1) Polycythemia hypertonica; no splenomegaly; blood pressure, 180 mm.; (2) splenomegalic polycythemia rubra, with tuberculosis or other splenic disease; (3) secondary polycythemia rubra, with splenomegaly and portal stasis, and (4) chronic cardiopulmonary cyanosis or Ayerza's disease.

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266. Gregg, H. W.; Lutz, B. R., and Schneider, E. C.: Am. J. Physiol. **50:** 216 (Nov.) 1919.
267. Kemp, G. T.: Am. J. Physiol. **10:**33, 1903-1904.
268. Thaysen, T. E. H.: Ugesk. f. Læger **82:**473 and 514 (April 8 and 15) 1920.
269. Editorial, J. A. M. A. **74:**1781 (June 26) 1920.
270. Lamson, P. D.: J. Pharmacol. & Exper. Therap. **16:**125 (Sept.) 1920.
271. Bing, H. I.: Ugesk. f. Læger **82:**337 (March 11) 1920.
272. Weber, F. P.: Brit. M. J. **2:**658 (Oct. 30) 1920.

True polycythemia rubra in Herrenheiser's [273] case was an instance of increased production of red cells and reduced rate of destruction. Böttner [274] recommends irradiation of the long and short bones controlled by the blood picture. The roentgen-ray therapy of the spleen should be limited to irritative doses.

ANEMIAS

The question of the value of *iron in the treatment of anemias* is still under controversy. Nageli [275] thinks that in proper dosage iron elicits a strong bone marrow reaction throwing quantities of young red cells into the blood stream. Alder [276] says the improvement with Blaud's pills reaches its acme in three weeks, and then remains stationary. Hooper, Robscheit and Whipple [277] show that Blaud's pills have no influence in simple secondary anemias even when combined with varied diets.[278] Of the diets of value in secondary anemia, cooked lean beef heart and cooked liver are important for blood regeneration in dogs. Water extracts of meat and liver are not so effective.[279] Bread and milk diet does not give complete regeneration after hemorrhage, while the increase in red blood cells and hemoglobin is greater in a fasting animal than in one fed on sugar.[280]

Larrabee [281] considers *aplastic anemia* a type of an anemia group. Most benign anemias are due to an increased destruction or loss of blood cells, to compensate which some of the yellow bone marrow changes to red marrow, produces new corpuscles and then returns to the normal adult condition. In pernicious anemia there is intense marrow hyperplasia which still is not sufficient to balance the destruction. In aplastic anemia the marrow does not produce even the normal amount of blood cells and there is no compensatory hyperplasia. In aplastic anemia the power to produce red corpuscles, myelocytic leukocytes and platelets is all involved. The platelet decrease and prothrombin decrease explains the tendency to hemorrhage. Purpura hemorrhagica is probably an incomplete form of aplastic anemia, only the platelet producing power being affected. Mensi [282] adds to the cases of

273. Herrnheiser, G.: Deutsch. Arch. f. klin. Med. **130**:315 (Oct. 24) 1920.
274. Böttner, A.: Deutsch. med. Wchnschr. **46**:66 (Jan. 15) 1920.
275. Nägeli: Schweiz. med. Wchnschr. **50**:661 (July 29) 1920; Abstr. J. A. M. A. **75**:966 (Oct. 2) 1920.
276. Alder, A.: Ibid., p. 663.
277. Hooper, C. W.; Robscheit, F. S., and Whipple, G. H.: Am. J. Physiol. **53**:263 (Sept.) 1920.
278. Whipple, G. H.; Hooper, C. W., and Robscheit, F. S.: Am. J. Physiol. **53**:151 (Sept.) 1920
279. Hooper, C. W.; Robscheit, F. S., and Whipple, G. H.: Ibid., p. 206.
280. Whipple, G. H.; Robscheit, F. S., and Hooper, C. W.: Ibid., p. 286.
281. Larrabee, R. C.: J. A. M. A. **75**:1630 (Dec. 11) 1920.
282. Mensi, E.: Pediatria, Naples **28**:785 (Sept.) 1920.

pernicious anemia in children reported in the literature, making a total of twenty-nine cases. The etiology is obscure after the elimination of a few cases due to syphilis, tuberculosis and intestinal parasites. Meulengracht [283] reports *familial pernicious anemia* developing in four sons and the son of one daughter. This familial occurrence confirms the assumption of one or more endogenous factors.

Kaznelson [284] cites a case of *hemolytic anemia* dating from childhood, in which splenectomy ended the symptoms but did not eradicate the disease. Fraginoli [285] studied nine cases of hemolytic jaundice. The characteristic feature is the presence of numerous nucleated red corpuscles, megaloblasts, without sufficient anemia to account for them. This, he considers, is due to the persistence of megaloblast-producing tissue which normally undergoes involution soon after birth. The persistence of this one element throws the whole system out of gear. Clark and Evans [286] suggest that the reason why hemolysis does not occur in normal blood is because the serum has a protective power against the natural hemolysins in the blood. If this protective power be diminished the normal hemolytic substances may act without any increase in the hemolysin or the introduction of foreign hemolysins. Ordinarily, the protective power is a constant, but in the anemias, especially with splenic involvement, the protective power is diminished.

Hallez [287] reports two cases of anemia with splenomegaly in infants. Tuohy's [288] case was associated with a leukocytosis of 65,000, 99 per cent. of the leukocytes being polymorphonuclears. In embryos up to the fourth month both spleen and bone produce red blood cells. After the fourth month, erythrocytes and myeloblasts are formed almost entirely from bone marrow and the spleen functions in blood destruction, the two hyperfunctioning coordinately. The method of leukocyte destruction and replacement is not known.

In hemolytic anemias, Speidel, Hoff and Nickson [289] suggest partial splenectomy. In dogs partial splenectomy is accompanied by distinct polycythemia. Biffis,[290] Losio [291] and Hartmann [292] report cases in

283. Meulengracht, E.: Ugesk. f. Læger **82**:777 (June 17) 1920; Abstr. J. A. M. A. **75**:644 (Aug. 28) 1920.
284. Kaznelson, P.: Wien. Arch. f. inn. Med. **1**:563 (Aug.) 1920.
285. Fraginoli, A.: Policlinico, Rome **27**:337 (Sept.) 1920.
286. Clark, H. M., and Evans, F. A.: Bull. Johns Hopkins Hosp. **31**:354 (Oct.) 1920.
287. Hallez, G. L.: Nourrisson, Paris **8**:154 (May) 1920.
288. Tuohy, E. L.: Am. J. M. Sc. **60**:18 (July) 1920.
289. Speidel, W. C.; Hoff, E., and Nickson, D. H.: Northwest Med. **19**:94 (April) 1920.
290. Biffis, P.: Policlinico **26**:393 (Nov.) 1919.
291. Losio, L.: Ibid., p. 410.
292. Hartmann, H.: Bull. de l'Acad. de méd. Par. **83**:599 (June 29) 1920; Abstr. J. A. M. A. **75**:571 (Aug. 21) 1920.

which the spleen was removed. Cushing and MacCallum [293] report two cases of splenic anemia with splenectomy in 1898. So little has been added to our knowledge of the pathology of the spleen that MacCallum's report in 1898 is included unchanged. The blood changes after removal of the spleen in a case without anemia, were studied by Hall.[294] The leukocyte count was increased for three months with no eosinophilia. The Arneth index suggests that the increase is due, in part, to the removal of some factor restricting the production of white corpuscles. Henn [295] finds in experimental animals that the corpuscles of splenectomized dogs are more strongly resistant to hypotonic sodium chlorid solutions than in control animals; and in young animals the coagulation time is hastened. Downs and Eddy [296] find that the subcutaneous injection of free splenic extract is followed immediately by a temporary decrease in the number of the red blood cells.

Sappington [297] reports another case of Gaucher's disease bringing the total of authentic cases with histologic examinations to twenty-five cases.

THE LEUKOCYTES

Loeper [298] studied the leukocytosis in animals after the injection of epinephrin. The mononuclear leukocytes seem to share in the production of antibodies, and increasing numbers presage recovery and immunity. Sprunt and Evans [299] report six cases of acute tonsillitis, infections of the upper respiratory tract and moderate general lymphadenitis in which there was a mononuclear leukocytosis. Audain [300] says that invading infections are combated by polymorphonuclear leukocytes when the region invaded is poor in lymphoid elements; but when lymphoid elements are present, they assume the local defense and mononucleosis is the rule. With infections of the lungs and bladder there is a polynucleosis throughout. Infections of the intestine, glands and spleen show a mononucleosis. If the lymphoid elements are not equal to the task, it is begun by the polymorphonuclears until the mononuclears become adequate for the defense. Ruppanner [301] finds that persons coming to the mountains have a pronounced leukocytosis at first, an acclimation leukocytosis, which yields in about six weeks to the moun-

293. Cushing, H., and MacCallum, W. G.: Arch. of Surg. **1**:1 (July) 1920.
294. Hall, M. W.: Am. J. M. Sc. **160**:72 (July) 1920.
295. Henn, C. S.: Am. J. Physiol. **52**:562 (July) 1920.
296. Downs, A. W., and Eddy, N. B.: Am. J. Physiol. **51**:279 (March) 1920.
297. Sappington, S. W.: J. A. M. A. **75**:105 (July 10) 1920.
298. Loeper, M.: Progrès méd. **35**:37 (Jan. 25) 1920.
299. Sprunt, T. P., and Evans, F. A.: Bull. Johns Hopkins Hosp. **31**:410 (Nov.) 1920.
300. Audain, L.: Presse méd. **28**:796 (Nov. 6) 1920.
301. Ruppanner, E.: Schweiz. med. Wchnschr. **50**:105 (Feb. 5) 1920.

tain leukocyte picture, i.e. a total count at the low range of normal, with the percentage of lymphocytes above normal and the neutrophil percentage below normal.

Otani [302] finds an increased phagocytic power for the particular species of invading organisms. The acceleration may be an immunologic reaction, making an early diagnosis of tuberculosis, typhoid and dysentery possible. Tunnicliff [303] finds that injections of leukocyte extracts produce an increase in the number and in the activity of the leukocytes.

Schilling [304] thinks the number of nuclear lobes in the neutrophil polymorphonuclears is as important as the pulse in examinations of the heart. Displacement to the left in the Arneth classification is a constant clinical symptom in some infections. Friedburg [305] gets no effect on the white blood corpuscles by stimulation of the vegetative nervous system in children. There is no vagotonic or sympatheticotonic blood picture. The eosinophils have a tendency to parallel the lymphocyte variations, and the mononuclears parallel the polymorphonuclears.

Giffin [306] reports a persistent eosinophilia of 73.6 per cent. in a case with a leukocytosis of 21,800 and splenomegaly. Removal of the spleen increased the eosinophils to 90.7 per cent. and the leukocytes to 211,000. A special function of the spleen with respect to eosinophil cells or with respect to the toxins the eosinophils absorb is suggested. Klinkert [307] considers eosinophilia a sign of a constitutionally inferior autonomic nervous system entailing a predisposition to asthma, gout, urticaria and migraine.

Pepper [308] diagnosed a case of typhoid fever on the finding of numerous cells resembling endothelial leukocytes in the urine. Tryon [309] gives the leukocyte reactions in paratyphoid dysentery and after vaccination. The differential counts are not so variable as the total counts, and in all cases the platelet count was high. Ingles [310] finds a definite but variable *leukocytosis in trench fever*. Both polymorphonuclears and nongranular cells are increased, the nongranular cells being the more numerous which may be a factor in differential diagnosis of trench fever.

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302. Otani, M.: Med. Rec. **97**:439 (March 13) 1920.
303. Tunnicliff, R.: J. Infect. Dis. **26**:447 (May) 1920.
304. Schilling, V.: Ztschr. f. klin. Med. **89**:1, 1920.
305. Friedburg, E.: Monatschr. f. Kinderh. **18**:432 (Aug.) 1920.
306. Giffin, H. Z.: Am. J. M. Sc. **158**:618 (Nov.) 1919.
307. Klinkert, D.: Ztschr. f. klin. Med. **89**:143, 1920.
308. Pepper, O. H. P.: Am. J. M. Sc. **160**:336 (Sept.) 1920.
309. Tryon, G.: Boston M. & S. J. **183**:643 (Dec.) 1920.
310. Ingles, W. K.: Med. J. Australia **2**:431 and 454, 1919.

Canelli [311] calls attention to the fact that azurophilia is not a blood finding that is specific for measles, being also found in pneumonia and scarlet fever; but the *azurophilia in measles* is so marked and pronounced that it may aid in differential diagnosis.

Schilling [312] reports two cases of ulcerative endocarditis giving very high mononuclear leukocyte counts. Rosenow [313] considers the leukopenia of influenza due to the peculiar properties of the streptococci or other organisms associated with the disease.

Escomel [314] examines blood for bacteria before any surgical operations. The blood is mixed with 15 c.c. of a 1 per cent. acetic acid solution, centrifuged and the supernatent fluid poured off. The sediment is washed with physiologic serum, and stained smears made from the sediment show leukocytes, bacteria and parasites.

LEUKEMIA

Decastello [315] calls attention to the occurrence of *Bence-Jones proteinuria* in leukemia. It seems to originate in a pathologic condition of the bone marrow. Howell [316] finds that six of eight patients with leukemia produced no agglutinins after injection of typhoid vaccine. This suggests that in leukemia the power to produce anticellular antibodies also resides in the hematopoietic tissue.

Tancre [317] reports a case of acute lymphatic leukemia developing after a suppurating process at the umbilicus. Ward [318] considers the preleukemic states in infancy, referring to the large series of cases of Von Jaksch's disease reported by Martelli.[319] The blood may show a myeloid blood picture with slight leukocyte increase, a few myelocytes and anomalous cells, Tuerck cells, plasma cells and large mononuclears, and perhaps a few nucleated red cells, and a color index of over one. Gunewardene [320] followed the development of such a case through to a definite myelogenous leukemia picture.

Fitz [321] reports the simultaneous occurrence of diabetes and myelogenous leukemia in one patient, which he believes are independent of each other. Ravenna [322] cites three cases of *myelogenous leukemia*

311. Canelli, A. F.: Rev. di clin. Pediatrica **18**:82 (Feb.) 1920.
312. Schilling, V.: Ztschr. f. klin. Med. **88**: 1919.
313. Rosenow, E. C.: J. Infect. Dis. **26**:492 (June) 1920.
314. Escomel, E.: Anales de la Faculdad de med. de Lima **3**:12 (July-Aug.) 1920; Abstr. J. A. M. A. **75**:1601 (Dec. 4) 1920.
315. Decastello, A.: Wien. Arch. f. inn. Med. **1**:335 (June 1) 1920.
316. Howell, K. M.: Arch. Int. Med. **26**:706 (Dec.) 1920.
317. Tancré, E.: Arch. f. Kinderh. **67**:7 (Nov. 30) 1918.
318. Ward, G.: Brit. J. Child. Dis. **17**:18 (Jan.-March) 1920.
319. Martelli, C.: Gazz. Internaz. di Med., 1919. Quoted from Ward.
320. Gunewardene, T. H.: Brit. J. Child. Dis. **17**:9 (Jan.-March) 1920.
321. Fitz, R.: J. A. M. A. **75**:1331 (Nov. 13) 1920.
322. Ravenna, F.: Riforma med. **36**:86 (Jan. 24) 1920.

systematically treated with benzol. The effect is purely symptomatic and the symptoms return in a more grave form. Lafleur's [323] patient improved under the combination of benzene and Roentgen-ray treatment.

THE BLOOD PLATELETS

Marchesini [324] thinks platelets result from the debris of unstable red corpuscles. The process of platelet formation may be watched in the large erythrocytes of frogs and birds. Mallory and Medlar [325] find interesting changes in blood plates under the dark field microscope. At first, the platelets are round or ovoid, then they elongate into threads and star-shaped bodies with threadlike projections showing serpentine movement. In stained preparations these platelets give the reaction of fibrin.

Gram [326] uses the Thomsen method of obtaining a hemogeneous suspension of platelets in plasma, and then dilutes 1:20 with a 2 per cent. solution of liquor formaldehyd in 0.9 per cent. sodium chlorid solution and counts direct in a Thoma Zeiss chamber.[327] This gives the number of platelets per c. mm. of citrated plasma. This figure is multiplied by constants which are determined by the hemoglobin content to find the number of plates in whole blood. The normal count is between 300,000 and 450,000. Pernicious anemia with a platelet count of under 100,000 tends to terminate fatally without remissions. Jost [328] finds that platelet extract by the mouth shortens the coagulation time and tends to arrest hemorrhage.

THE COAGULATION OF THE BLOOD

Several new methods for determining the coagulation time of blood are given. Gram [329] finds that too low recalcinations of citrated plasma lengthen the coagulation time. He uses the sedimentation plasma from the Thomsen method for counting blood plates because in the centrifugalized specimen the blood platelets are thrown down with the reds, and the coagulation time is doubled in normal bloods but not in bloods where the platelets are deficient. In hemophilia, the coagulation time is lengthened with a normal platelet count. This may be used for the diagnosis of the hemorrhagic diathesis. Love [330] determines the coagu-

323. Lafleur, H. A.: Canad. M. A. J. **10**:996 (Nov.) 1920.
324. Marchesini, R.: Policlinico **27**:227 (Feb. 23) 1920.
325. Mallory, F. B., and Medlar, E. M.: J. M. Res. **41**:327, 1920.
326. Gram, H. C.: Arch. Int. Med. **25**:325 (March) 1920.
327. Gram, H. C.: Ugesk. f. Læger **82**:718 (June 3) 1920; Abstr. J. A. M. A. **75**:282 (July 24) 1920.
328. Jost, W.: Cor.-Bl. f. schweiz. Aerzte **49**:1909 (Dec. 11) 1919.
329. Gram, H. C.: Bull. Johns Hopkins Hosp. **31**:364 (Oct.) 1920.
330. Love, G. R.: Med. Rec. **98**:436 (Sept.) 1920.

lation time by means of capillary tubes. The blood is drawn into paraffined test tubes and within two minutes is sucked up in capillary tubes and the time taken. Two minutes before the normal coagulation time, 2 cm. of the first tube is broken off drawing the fragments gently apart to avoid injuring the fibrin thread. Other segments of tube are broken off until the time interval is such that the fibrin thread will stretch from 7 to 10 mm. across the break. This is the end point. Rodda [331] uses a drop method, the drops from the heel of the baby being received in a clean watch glass containing a No. 6 shot. A second watch glass is inverted over the first. The crystal is tilted every thirty seconds, until the shot no longer rolls. The average clotting time for a new-born baby is seven minutes with a range of from five and one-half to eight and one-half minutes. King and Murray [332] draw the blood into open tubes with attachment below for a stop cock and a Luer needle. Two c.c. are drawn into the tube direct from the vein and kept at a temperature of from 65 to 90 F. The coagulation time is the nearest half minute from the instant the blood first appears to the moment when the tube may be inverted without displacing the clot.

The *coagulation time* in embryonic blood of the pig, according to Emmel, Levinson and Fisch,[333] is six or eight times that of the adult, although the platelet count is the same. Platelet material from the adult and calcium chlorid both reduce the coagulation time, but not as much as the addition of tissue extract which makes the coagulation time the same as the adult. Stephan [334] studies the active and inactivated blood serum and blood plasma of the specimen to be tested with the action of normal serums and plasms; and the effect of normal blood serum and blood plasma of a known value on the coagulation time of the unknown specimen. This gives the pathologic factors. With his method, the coagulation time of normal blood is from twenty-eight to thirty-three minutes.

Corachan and Mones [335] insist on the value of the *warning of the coagulation index* as a pre-operative test. When the coagulation time is slow 3 or 4 grains of calcium chlorid per day, before the operation, may bring the coagulation time up to normal. Petrén [336] tests coagulation of blood in jaundice by the bile-acid-alkali test. Normally from 0.55 to 0.66 per cent. of bile-acid-alkali is necessary to prevent coagulation. If

331. Rodda, F. C.: Am. J. Dis. Child. **19**:269 (April) 1920.
332. King, G., and Murray, H. A.: J. A. M. A. **74**:1452 (May 22) 1920.
333. Emmel, V. E.; Levinson, S. A., and Fisch, M. E.: J. Exper. M. **31**: 117 (Feb.) 1920.
334. Stephan, R.: Deutsch. med. Wchnschr. **46**:684 (June 17) 1920.
335. Corachan, M., and Mones, F. G.: Siglo méd., Madrid **66**:935 and 960 (Nov. 1) 1919; Abstr. J. A. M. A. **74**:68 (Jan. 3) 1920.
336. Petrén, G.: Beitr. z. klin. chir. **120**:501, 1920.

from 0.45 to 0.40 per cent. prevents coagulation, there is danger of hemorrhage during or after operation; if 0.30 per cent. prevents hemorrhage, operations are contraindicted.

Kinsella and Brown [337] report a retarded coagulation time and a decrease in the platelet count in influenza.

Szenes [338] finds that after an initial retardation, *irradiation of the spleen* accelerates the coagulation time, doubtless due to the breaking down of lymphocytes in the splenic follicles liberating thromboplastic substances. Jurasz [339] thinks irradiation of the spleen with roentgen rays may prove effective in stopping hemorrhage, and be of value from twelve to fifteen hours before operation in cases in which the coagulation time is lengthened. Stephan [340] has used this method successfully in a case of purpura fulminans.

Mills [341] finds that extract of the lung and kidney, and to a lesser degree extracts of skin, possess a strong coagulative activity. The potency of the lung may be a special protection in pulmonary diseases. Tait [342] thinks that the arrest of hemorrhage is due to the agglutination of certain cells "thigmocytes" which are highly phagocytic and on breaking down exude thrombin. When anticoagulants are added, the thigmocytes (platelets and spindle cells) are unaltered. Foreign surfaces and dirt give surfaces to which the cells may adhere, and become altered. Houssay and Sordelli [343] find that twenty-two venoms, including cobra venom, prevent coagulation by destroying cytozym which normally combines with serozym and calcium to form thrombin, but that lachesis and crotalus venoms have a special coagulating power that coagulates even citrated blood. Menten [344] finds that the anticoagulant power of hirudin and cobra venom is destroyed by an increase in acidity, while with peptone prevention the acidity is always increased, so that the mechanism differs with peptone.

The relationship between *coagulation time in the blood of the newborn and cerebral hemorrhage* becomes increasingly important with the discovery that cerebral hemorrhage is found in more than 50 per cent. of all necropsies on infants dying during the first few days of life. Rodda [345] finds that coagulation time and bleeding time are prolonged from the first to the fifth day of life, with the maximal retardation on

337. Kinsella, R. A., and Brown, G. O.: J. A. M. A. **74**:1070 (April 17) 1920.
338. Szenes, A.: München. med. Wchnschr. **67**:786 (July 2) 1920.
339. Jurasz, A. T.: Zentralbl. f. Chir. **47**:824 (July 3) 1920.
340. Stephan, R.: München. med. Wchnschr. **67**:309 (March 12) 1920.
341. Mills, C. A.: J. Biol. Chem. **40**:425 (Dec.) 1919.
342. Tait, J.: J. Physiol. **53**:19 (Sept. 5) 1919.
343. Houssay, B. A., and Sordelli, A.: Prensa méd. Argentina **6**:113 (Oct. 20) 1919; Abstr. J. A. M. A. **74**:140 (Jan. 10) 1920.
344. Menten, M. L.: J. Biol. Chem. **43**:381 (Sept.) 1920.
345. Rodda, F. C.: J. A. M. A. **75**:452 (Aug. 14) 1920.

the fifth day and a return to normal on the tenth day. The delayed times correspond to the age incident of hemorrhagic disease and cerebral hemorrhage. The coagulation time may be brought back to normal by the subcutaneous injection of blood—which Voncken [346] calls homohemotherapy. Foote [347] says that blood coagulation tests should be routine examinations on all newborn infants, and in every case of suspected intracranial hemorrhage substances to increase blood coagulability are indicated.

HEMOPHILIA

Nobecourt [348] says that the most effective treatment for hemophilia at present is horse serum and the subcutaneous injection of peptone solutions. He gives the treatment in detail. It gives marked improvement but is not a cure. After transfusion in Bulger's [349] case of hemophilia, the coagulation time was the same as in the control, but both the coagulation time and the prothrombin time lengthened, but at the end of a month it was still less than before the transfusion. Weil [350] controls his hemophilia cases by subcutaneous injection of 20 c.c. of animal or human blood serum every two months. Of fifty cases treated in fifteen years he has not lost one patient. He regards hemophilia as a congenital functional malformation of the elements in which blood and blood vessels originate. Chalier [351] considers the mother's blood serum the most effectuál treatment for congenital hemophilia.[352]

INTRACARDIAL AND INTRAVENOUS INJECTIONS

The controversy concerning the best method for *blood transfusion* is still unsettled, direct and indirect methods having their advocates. Schaaf [353] says that the citrate method is the one of choice, and in pernicious anemia transfusion produces an immediate general improvement, but it has no curative value. Graham [354] says that the ideal method is either direct from artery to vein or indirect from vein to vein without citrate. In pernicious anemia transfusion may cause remissions. The benefit is not necessarily in proportion to the volume of blood received. Terrien,[355] instead of transfusing an infant 13 months

346. Voncken, J.: J. A. M. A. **75**:307 (July 31) 1920.
347. Foote, J. A.: Am. J. Dis. Child. **20**:18 (July) 1920.
348. Nobecourt, P.: Paris méd. **9**:452 (Dec. 6) 1919.
349. Bulger, H. A.: J. Lab. & Clin. M. **6**:102 (Nov.) 1920.
350. Weil, P. E.: Médecine, Paris **1**:354 (March) 1920.
351. Chalier, J.: Rev. de méd. **36**:522 (Sept.-Oct.) 1919.
352. Chalier, J.: Presse méd. **28**:773 (Oct. 30) 1920.
353. Schaaf, F. H. K.: Minnesota Med. **3**:382 (Aug.) 1920.
354. Graham, J. M.: Edinburgh M. J. **24**:282 (May) 1920.
355. Terrien, E.: Bull. et mém. soc. méd. d. hôp. de Par. **43**:1134 (Dec. 26) 1919.

old with malignant measles, injected whole blood subcutaneously with excellent results.

Van Dijk [356] calls attention to the development of malaria in a patient who was given serum from an influenza convalescent. The donor's blood contained no parasites at the time of transfusion although they had been present some months previous. Busman [357] calls attention to the fact new rubber tubing may give a marked reaction when it is used for transfusion. It is rendered harmless by soaking for six hours in normal sodium hydrate solution. Widakowich and Madrid [358] note that agglutination reactions in serum and in plasma differ. Normally, serum agglutinates alien erythrocytes more intensely than plasma, but in syphilitics, plasma agglutinates more than serum, and this is of importance in blood grouping for transfusion into a syphilitic. Kolmer and Matsumoto [359] point out that while all horse serum contains agglutinins for human erythrocytes, intravenous injection of horse serum does not introduce sufficient agglutinins and hemolysins to cause effects referable to intravascular agglutination.

Henschen [360] has succeeded in permanently *resuscitating the heart* after complete arrest by the direct injection of a stimulant. He injects epinephrin and pituitary extract directly into the left ventricle. In four cases the effect was instantaneous, but in only one case was recovery complete and permanent. Henschen thinks this is the only successful case on record. He gives illustrations showing five danger zones to be avoided in intracardial injections. Vogeler [361] injected epinephrin directly into a heart which had ceased to beat, and in half a minute the heart was beating and respiration began, to stop again in about six minutes; a second injection had the same transient effect and the child died. Heydtoff [362] injected epinephrin into the heart in a case in which there was total arrest of the heart for five or six minutes, and the heart began to beat rapidly. Artificial respiration was necessary for a time. This is a second case with recovery.

Aikman [363] discusses the methods for administration of saline and other solutions to children. In intravenous injections, one-sixtieth of the body weight can be introduced. In infants this is best done through

356. Van Dijk, H.: Nederlandsch Tijdschr. v. Geneesk. **2**:1181 (Sept. 25) 1920.
357. Busman, C. J.: J. Lab. & Clin. M. **5**:693 (Aug.) 1920.
358. Widakowich, V., and de Madrid, S.: Semana méd. **27**:296 (Feb. 26) 1920.
359. Kolmer, J. A., and Matsumoto, M.: J. Immunol. **5**:75 (March) 1920.
360. Henschen, K.: Schweiz. med. Wchnschr. **50**:261 (April 1) 1920; Abstr. J. A. M. A. **74**:1610 (June 5) 1920.
361. Vogeler, K.: Deutsch. med. Wchnschr. **46**:740 (July 1) 1920.
362. Heydtoff, E.: Monatschr. f. Geburtsh. u. Gynäk. **51**:318 (May) 1920.
363. Aikman, J.: J. A. M. A. **74**:244 (Jan. 24) 1920.

the fontanel, but with a closed fontanel either the jugular vein or the femoral vein should be used. Wentworth's technic for entering the femoral vein is given in detail. Friedmann [364] is using the drop method for intravenous infusion. When there is increased lacrimal secretion or slight edema of the eyelids, it is a sign that the patient is getting too much fluid.

Pfalz [365] uses glucose injections in heart disease when there is an indication that the heart is not sufficiently nourished. John [366] uses intravenous injection of glucose solutions in pneumonia. None of the sugar appears in the urine and it all seems to be used for nutrition. Richaud [367] points out that sugar solutions are less toxic than saline solutions and as much as a 1,300 c.c. of isotonic glucose, saccharose or lactose solution may be injected into the veins. Sugar tones up the heart and has an antitoxic action. Rathery and Boucheron [368] inject hypertonic sugar solutions (30 per cent. glucose) intravenously to stimulate diuresis in selected cases of nephritis. It is contraindicated in chronic nephritis with "azotemia." Wells and Blankinship [369] use the intravenous injection of hypertonic glucose solutions in influenzal pneumonia. It withdraws fluid from the body tissues to keep the blood isotonic, and the sugar content of the blood becomes normal without any glycosuria. Barthélemy [370] uses intravenous injections of blood plasma and gum solutions. In cases in which blood pressure must be raised and sustained, gum saline solutions are especially indicated. Bayliss [371] says that gum acacia solutions (6 or 7 per cent. in 0.9 per cent. sodium chlorid solution) are capable of replacing lost blood unless the amount lost is more than 75 per cent. of the blood volume. It does not produce anaphylaxis, or hemolysis nor does it agglutinate blood corpuscles in man.

INTRAVENOUS THERAPY IN SHOCK

Bayliss [371] believes shock is due to the absorption of toxic substances produced by injured cells. Dale [372] finds that exhaustion, pain, cold, thirst, hemorrhage, anesthetics and histamin all have been alleged to play a part in shock. Widal, Abrami and Brissaud [373] believe shock

364. Friedmann, M.: Deutsch. Ztschr. f. Chir. **151**:352 (Nov.) 1919.
365. Pfalz, W.: Deutsch. med. Wchnschr. **45**:1181 (Oct. 23) 1919.
366. John, H. J.: Am. J. M. Sc. **160**:542 (Oct.) 1920.
367. Richaud, A.: Paris méd. **10**:53 (July 17) 1920.
368. Rathery, F., and Boucheron, H.: Bull. et mém. Soc. méd. d. hôp. de Par. **44**:61 (Jan. 16) 1920.
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is anaphylactic in its nature and due to a destruction of colloids. The most recent views on shock are summed up in two editorials in the *Journal of the American Medical Association.*[374] Anything that causes a diminished volume of the blood that is in effective circulation attributable to loss of normal capillary tone, or to morbid permeability of the capillary walls is followed by a shocklike collapse.[375] Gasser, Erlanger and Meek,[376] in a series of studies on secondary shock, find the blood volume decreased in all experimental cases, and in treatment they conclude that the restoration of blood volume through osmotic action from the tissues is more efficacious than injecting the full amount of the fluid. Mann [377] believes that colloid solutions are better than isotonic salt solutions for restoring blood volume but not as good as blood serum.

The most interesting development in intravenous therapy is the use of strongly hypertonic solutions. White and Erlanger [378] point out that strongly hypertonic glucose and gum acacia solutions injected intravenously cause an immediate increase in blood volume. The crystalloids attract the fluids more rapidly, and the colloids tend to hold the fluid in the circulatory system for a longer period of time. The old rules requiring an absolutely isotonic solution for intravenous injection are swept to the winds by the report of Sachs and Belcher [379] who gave 100 c.c. of a saturated solution of sodium chlorid intravenous at the rate of 1 c.c. per minute. The injection was repeated three successive times. This was a case of brain tumor in which the intracranial pressure was too high for a successful operation, and the saline injection reduced this pressure. The fragility of the red blood corpuscles on daily examinations never varied beyond the normal limits.

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